ARCHIVES OF OPHTHALMOLOGY

VOLUME 33 (old series Volume 90) APRIL 1945 NUMBER 4

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CHOICE OF OPERATION FOR PRIMARY GLAUCOMA COMBINED WITH CATARACT

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Choice of the initial operation for an eye with both primary glaucoma and cataract is a recurring problem which has had scant attention. Many ophthalmologists routinely remove the cataract first, with or without preliminary iridectomy, hoping to control the glaucoma at the same time. Further surgical treatment, usually cyclodialysis, is relied on should the tension again become elevated. Other ophthalmologists perform some operation for glaucoma first, believing that later cataract extraction offers the best chance for ultimate vision. A few ophthalmic surgeons prefer to perform an anterior sclerectomy or inclusion of an iris pillar at the time the cataract is extracted.

The literature contains no statistical evidence indicating which procedure gives the best final result in an eye with primary glaucoma and cataract. Elschnig [1] and van Lint [2] suggested that if the tension in such an eye never rises above 35 mm. of mercury and can be reduced to 20 mm. with miotics, cataract extraction should be the first operation; otherwise, cyclodialysis should be performed, followed in four weeks by extraction of the lens. Spaeth [3] states that preliminary iridectomy and subsequent cataract extraction should be done only if the glaucoma is clearly secondary to swelling of the lens. If there is any question that the glaucoma is primary and noninflammatory, he believes that it is always best to perform an anterior fistulizing operation first and to remove the cataract later. O'Brien [4] prefers to perform anterior sclerectomy at the time the cataract is extracted, and he has been pleased with his results. Kirby [5] believes that the intraocular tension should be as nearly normal as possible before cataract extraction. If the tension is above normal, he performs a fistulizing operation at least six weeks before cataract extraction. If the tension is still above normal, a second operation for glaucoma is performed prior to extraction. If the tension is normal at the time of extraction, Kirby avoids the filtering area by making a corneal section in front of the bleb; but if the fistulizing operation has resulted in actual hypotension (8 mm. of mercury or below), he makes his section through the filtering area, in the hope that as healing proceeds there may be some increase in tension.

The present study indicates the results obtained at the Wilmer Institute with various surgical procedures for different types of primary glaucoma with cataract.

MATERIAL

The case histories of all patients with the combination of primary glaucoma and cataract uncomplicated by other ocular disease who were operated on at the Wilmer Ophthalmological Institute between Jan. 1, 1927 and Jan. 1, 1944 were reviewed. The lenticular changes varied from opacities of such degree that it was apparent later extraction would be indicated to hypermature cataracts. The histories were discarded if the follow-up period was less than six months after cataract extraction if that was the only operation, or less than six months after the second procedure if an operation for glaucoma was also performed. There was no other basis for the selection of cases. Forty-four case histories met the requirements for this study. The average follow-up period was nineteen months. The average age of the patients was 68 years.

ANALYSIS OF RESULTS

In the 44 cases studied there were (1) 29 eyes with chronic noncongestive glaucoma, (2) 10 with chronic congestive glaucoma and (3) 5 with

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Read at the Forty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology, Chicago, Oct. 11, 1944.

1. Elschnig, A.: Augenärztliche Operationslehre, in Graefe, A., and Saemisch, T.: Handbuch der Augenheilkunde, ed. 3, Berlin, Julius Springer, 1922, vol. 2, p. 1240.

2. van Lint, A., in Bailliart, P., and others: Traité d'ophtalmologie, Paris, Masson & Cie, 1939, vol. 7, p. 677.

3. Spaeth, E. B.: The Principles and Practice of Ophthalmic Surgery, ed. 3, Philadelphia, Lea & Febiger, 1944, p. 593.

4. O'Brien, C. S.: Personal communication to the author.

5. Kirby, D. B.: Prevention and Handling of Complications Arising During and After Cataract Extraction, Arch. Ophth. **25**:866-901 (May) 1941.

acute congestive glaucoma. The eyes with chronic noncongestive glaucoma were subdivided into two groups: (*a*) 21 in which the glaucoma could not be controlled with miotics and (*b*) 8 in which the glaucoma could be controlled with miotics.

The glaucoma was eventually controlled in 39 of these eyes, while in 5 it progressed to absolute glaucoma in spite of operation. The efficacy of the various procedures used depended in large measure on the type and stage of the glaucoma.

Chronic Noncongestive Glaucoma not Controllable with Miotics.—The results of various operations in the 21 eyes with chronic noncongestive glaucoma not controllable with miotics are given in table 1. Fourteen of these eyes were subjected to an anterior fistulizing operation as the initial procedure, the cataract being extracted from three weeks to several years afterward. In 11 of these 14 eyes an initial trephination controlled the tension without miotics, and in all 11 the tension remained normal without miotics after subsequent cataract extraction. In 1 eye a Lagrange anterior sclerectomy controlled the tension without miotics; but after cataract extraction the tension again rose, although it could be controlled with pilocarpine. In another eye, trephination controlled the tension only with use of miotics, and in a third miotics were necessary after both iridencleisis and trephination. In the last 2 eyes the tension rose after subsequent cataract extraction but was controlled in both instances with a single cyclodialysis and pilocarpine. In these 14 eyes an initial fistulizing operation followed by cataract extraction gave satisfactory results, later miotics or cyclodialysis being required in only 3 instances.

In 6 eyes the initial operation was a combined cataract extraction, 3 intracapsular and 3 extracapsular extractions. In all these eyes the tension became elevated within six weeks and did not respond to miotics. A trephination controlled the tension in 1 of them, and a single cyclodialysis and pilocarpine, in 2 others. One eye was almost blind from glaucoma before cataract extraction and was not operated on again. Two others did not respond to cyclodialysis but progressed to absolute glaucoma.

The initial procedure in 1 eye was a combined extracapsular cataract extraction and anterior sclerectomy. Tension afterward remained normal, without miotics.

Thus, in cases of primary noncongestive glaucoma with cataract, when the glaucoma was uncontrolled with miotics, an initial fistulizing operation prior to the extraction of the cataract

TABLE 1.—*Results of Operative Procedures, Including Cataract Extraction, in Twenty-One Cases of Primary Chronic Noncongestive Glaucoma Not Controllable with Miotics*

Operative Procedure	Total Number of Cases	Postoperative Results: Tension Controlled Without Miotics	Tension Controlled With Miotics	Tension Controlled After Additional Operation for Glaucoma	Tension Not Controlled
Anterior fistulizing operation and later cataract extraction	14	11	1	2	..
Combined cataract extraction	6	..	..	3	3
Combined cataract extraction with anterior sclerectomy (single procedure)	1	1	..	..	..
Total number of cases	21	12	1	5	3

TABLE 2.—*Results of Operative Procedures, Including Cataract Extraction, in Eight Cases of Primary Chronic Noncongestive Glaucoma Controlled with Miotics*

Operative Procedure	Total Number of Cases	Postoperative Results: Tension Controlled Without Miotics	Tension Controlled With Miotics	Tension Controlled After Additional Operation for Glaucoma	Tension Not Controlled
Combined cataract extraction	7	4	2	1	..
Combined cataract extraction with inclusion of iris pillar	1	..	..	..	1
Total number of cases	8	4	2	1	1

gave uniformly good results, while an initial cataract extraction in no instance controlled the tension and later operative procedures for the glaucoma were successful in only 3 instances.

Chronic Noncongestive Glaucoma Controlled with Miotics.—The results of various operative procedures in the 8 eyes of this type are shown in table 2.

In 7 of these eyes a combined cataract extraction was the initial procedure, the extraction being intracapsular in 6 and extracapsular in 1. Tension remained normal after operation without miotics in 4 eyes and with pilocarpine in 2 others. In the seventh eye a later trephination controlled the tension.

In the last eye of this group a combined intracapsular extraction with inclusion of an iris pillar was the initial procedure. The tension again became elevated within two weeks, and the patient did not submit to further operation.

Chronic Congestive Glaucoma.—The results of various operative procedures in 10 eyes with chronic congestive glaucoma and cataract are shown in table 3. The tension could not be controlled with miotics prior to operation in any of these eyes. In all there was a history or evidence of former attacks of low grade congestive glaucoma and the anterior chamber was shallow.

In 6 of these eyes an anterior fistulizing operation was performed first, the cataract being extracted in from ten days to four years later. In 2 the fistulizing operation controlled the glaucoma, and the tension remained normal without miotics after cataract extraction. In the third a trephination controlled the tension but it became elevated after subsequent cataract extraction, and later cyclodialysis was required to control the glaucoma. In 3 eyes the tension was not controlled by the initial fistulizing operation and remained elevated after later cataract extraction. It was eventually controlled in all these cases by additional operations for glaucoma.

In 4 of these eyes a combined cataract extraction (intracapsular in 2 and extracapsular in 2 cases) was the initial operation. The tension was normal thereafter in 3 without miotics. In the remaining eye the tension rose within two weeks, and vision was already so impaired from the glaucoma that further operative intervention was not thought justifiable.

Acute Congestive Glaucoma.—The results of the various operative procedures in 5 eyes with acute congestive glaucoma and cataract are shown in table 4.

In 2 eyes combined extracapsular cataract extractions were performed as the initial procedure because it was thought that the lens was

TABLE 3.—*Results of Operative Procedures, Including Cataract Extraction, in Ten Cases of Primary Congestive Glaucoma Not Controlled with Miotics*

Operative Procedure	Total Number of Cases	Postoperative Results: Tension Controlled Without Miotics	Tension Controlled With Miotics	Tension Controlled After Additional Operation for Glaucoma	Tension Not Controlled
Anterior fistulizing operation and later cataract extraction	6	2	..	4	..
Combined cataract extraction	4	3	..	..	1
Total number of cases	10	5	..	4	1

TABLE 4.—*Results of Operative Procedures, Including Cataract Extraction, in Five Cases of Primary Acute Congestive Glaucoma Not Controlled with Miotics*

Operative Procedure	Total Number of Cases	Postoperative Results: Tension Controlled Without Miotics	Tension Controlled With Miotics	Tension Controlled After Subsequent Operation for Glaucoma	Tension Not Controlled
Anterior fistulizing operation and later cataract extraction	1	1	..	..	..
Combined cataract extraction	2	2	..	..	..
Iridectomy with later cataract extraction	2	2	..	..	..
Total number of cases	5	5	..	..	..

swollen and was a contributing cause of the glaucoma. In both instances the tension was normal thereafter without miotics. In 2 eyes iridectomy controlled the tension without miotics, and in the fifth eye iridencleisis achieved this result. Subsequent cataract extraction was not followed by an increase in tension in any of these eyes.

COMMENT

This series of cases is too small to permit definite conclusions, but it does indicate the general reaction of eyes with different types of glaucoma complicated by cataract to various operative approaches.

If the glaucoma is noncongestive and cannot be controlled with miotics, an anterior fistulizing operation with subsequent cataract extraction appears to give the best results. Thus, the glaucoma was controlled in all 14 eyes treated in this way, and in 11 of them miotics were not needed. Of 6 eyes in which cataract extraction was the initial operation, the tension was controlled in 3 only after subsequent operation for glaucoma and was never controlled in the other 3 eyes.

If the glaucoma is noncongestive and can be controlled with miotics, a combined cataract extraction will usually control the glaucoma, and the eye need be subjected to only one operation. Extraction, therefore, seems indicated as the initial procedure for this type of glaucoma.

In eyes with chronic congestive glaucoma a combined cataract extraction as the initial procedure offers at least as much chance of controlling the tension as, and probably more than, an anterior fistulizing operation performed first.

In eyes with acute congestive glaucoma it seems best to do an iridectomy followed by cataract extraction or a combined cataract extraction if the tension is not too high or if the lens appears to be greatly swollen.

I have hesitated to perform an anterior fistulizing operation at the time of cataract extraction because of the delay in reformation of the anterior chamber and the possibility of the development of extensive anterior synechias. Therefore, no evaluation of this surgical procedure is possible in this series.

An interesting incidental observation was that making an incision for cataract through an anterior fistulizing bleb had no apparent bad effect. In 16 of the eyes in this series an anterior fistulizing operation was performed and controlled the tension without miotics prior to cataract extraction. A Graefe section was made through the bleb in 9 of these eyes, and a subsequent operation for glaucoma was necessary in only 1 instance. The section was made in front of the bleb in 7 eyes, and a subsequent operation for glaucoma was necessary in 1 eye. A study of the eyes in which a section was made through the filtering area showed that such a procedure did not necessarily, or even usually, reduce filtration appreciably, but theoretically filtration should be reduced more by this procedure than by making the section in front of the bleb. It seems wise, therefore, to adhere to Kirby's[5] tenet that the section should be made through the bleb if actual hypotension is present and in front of the bleb if tension is within normal limits.

SUMMARY

Results of operative procedure in 44 eyes with primary glaucoma and also cataract are analyzed. The following conclusions are drawn:

1. If the glaucoma is noncongestive and cannot be controlled with miotics, an anterior fistulizing operation should be performed prior to cataract extraction.

2. If the glaucoma is noncongestive but can be controlled with miotics, or if it is chronic congestive, a combined cataract extraction should be the initial procedure and is likely to be the only operation necessary.

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RESECTION OF THE LEVATOR PALPEBRAE MUSCLE FOR PTOSIS

WITH ANATOMIC STUDIES

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From a review of the literature on surgical treatment for ptosis, one is forced to the conclusion that every operation devised for the cure of this condition has one or more disadvantages. This must necessarily be so because of the inherent nature of ptosis.

Ptosis may be defined as a drooping of the upper lid, from whatever cause. It may be acquired or congenital, unilateral or bilateral, partial or complete. Acquired ptosis is usually due to disease of the upper lid, to disease of the levator palpebrae muscle or to disease of the nervous system. Congenital ptosis is due to a developmental failure of the levator muscle or, according to some investigators, of the nucleus of the third nerve.

The 80 odd surgical procedures suggested for the cure of ptosis may be divided into three categories: first, utilization of the frontalis muscle to support the lid; second, employment of the superior rectus muscle to lift the lid, and third, shortening of the levator palpebrae muscle to enhance its lifting power. None of these surgical procedures gives entirely satisfactory results. The operations in which the frontalis muscle is utilized to support the lid produce an unnatural appearance of the lid. Those in which the result depends on formation of adhesions between the superior rectus muscle and the upper lid often produce hypotropia and lagophthalmos. Of the various surgical procedures proposed, those designed to enhance the lifting power of the levator muscle are to be preferred when this muscle is not completely paralyzed. The chief argument against resection of the levator palpebrae muscle arises from the fact that inadequate correction sometimes results. The purpose of this paper is to determine, if possible, the reasons for such failures.

OPERATIONS FOR RESECTION OF THE LEVATOR PALPEBRAE MUSCLE

All surgical procedures designed to increase the lifting power of the levator muscle consist in shortening this muscle, either by tucking its tendon or by excising a part of it. The operative approach may be through the conjunctiva or through the skin.

Resection of the Levator Muscle or Its Attachments Through the Conjunctiva.—Bowman was perhaps the first to perform this operation. His technic was recorded by Mr. Bader, the curator and registrar of the Royal London Ophthalmological Hospital, in a report for the year ending Sept. 25, 1857.[1] Six operations for ptosis had been done that year, 4 of which consisted in the "usual operation of removal of an oval piece of skin" from the upper lid. The report stated:

> In one case in which this operation had been done with an unsatisfactory result, Mr. Bowman applied a new plan—he everted the lid and excised the posterior or upper edge of the palpebral cartilage, with about half an inch (1 cm.) of the tendon of the levator palpebrae inserted into it. Before actual removal of the piece, very fine threads were passed so as to bring together the edges, and then secure a shortening of the tendon of the levator muscle, to the extent of three-quarters of an inch (1.9 cm.). It is to be expected that it will be permanently shortened, and be enabled to assist in raising the lid. The upper cul de sac of the conjunctiva is exposed to little movement, and wounds there readily heal. The lid was turned back to its natural position.

This is the first record of an attempt to correct ptosis by resection of the levator muscle. From the description of this operation it is certain that Müller's muscle was resected. Whether the tendon of the levator palpebrae muscle was also included in the resection is difficult to say, because of Bowman's failure to differentiate Müller's muscle and the tendon of the levator muscle. A careful search of Bowman's publications showed that he made no distinction between this tendon (aponeurosis) and Müller's muscle.

Wheeler[2] performed, but did not publish, a somewhat similar procedure for resection of

Candidate's thesis for membership in the American Ophthalmological Society, submitted May 29, 1944.

1. Bowman, W.: Rep. Roy. London Ophth. Hosp. **1**:34-35, 1859.

2. Wheeler, J. M., cited by Meek, R. E.: Applied Anatomy of the Eye: Its Relation to Ophthalmic Surgery, Arch. Ophth. **26**:494-513 (Sept.) 1941.

the levator muscle. His technic is essentially that described by Bowman, as follows: The lid is everted with an Ehrhardt clamp; an incision is made through the conjunctiva 1 mm. above the upper margin of the tarsus, and the conjunctiva is freed from Müller's muscle approximately 10 to 15 mm. upward. An incision is then made through the tarsus 2 or 3 mm. from the upper margin. Müller's muscle, which is attached to this narrow strip of tarsus, and presumably also the tendon of the levator muscle lying anterior, are then dissected backward by following the normal line of cleavage between the levator muscle and the posterior surface of the orbicularis muscle. Three double-armed silk sutures are then passed through the conjunctiva near its cut edge and through the levator muscle 10 to 12 mm. above the tarsus. The muscle is then excised anterior to the sutures, and the three double-armed sutures are passed through the upper border of the cut edge of the tarsus and then through the skin and finally tied over rubber pegs. A petrolatum dressing is applied, and the sutures are removed on the fourth or fifth day. In 1917 Maddox [3] modified the Bowman operation by exposing Müller's muscle through the conjunctiva, cauterizing it and then tucking it with three sutures, so that it would be permanently shortened.

In 1896 Wolff [4] described an operation for resection of Müller's muscle.[5] He double everted the lid, made a horizontal incision through the upper portion of the conjunctival cul-de-sac and exposed Müller's muscle by reflecting the conjunctiva downward. Vertical incisions, 10 to 12 mm. apart, were made through the muscle, which was lifted up on strabismus hooks. Two surgical gut sutures were passed through the muscle and tied. The muscle was cut below the sutures, which were passed through the upper border of the tarsus, tied and cut short. The amount of resection required was equal to the difference in width between the two palpebral fissures. The conjunctiva was closed by means of five surgical gut sutures.

In 1909 Blaskovics first described his operation for resection of the levator muscle through the conjunctiva. His latest procedure [6] consists in everting the lid, injecting 2 cc. of a 2 per cent solution of procaine hydrochloride containing epinephrine hydrochloride under the upper fornix of the conjunctiva and incising the latter 2 or 3 mm. above the upper border of the tarsus. Three double-armed sutures are placed through the cut edge of the conjunctiva, and the latter is dissected free from Müller's muscle with scissors, using the three sutures for traction. Three other sutures are placed through Müller's muscle and presumably also through the tendon of the levator muscle 2 to 3 mm. above the tarsal border, and the muscle is cut free from the tarsus. By traction on these fixation sutures the attachments of the levator muscle are freed from the conjunctiva, on one side, and the "pretarsal tissues," on the other, so that the levator muscle can be pulled out easily. The three double-armed sutures in the conjunctiva are then passed through the levator muscle "at the correct height," and the muscle is excised distal to the sutures. The lid is now turned back with the cutaneous surface anterior; the lid is retracted upward to expose the levator muscle, and three mattress sutures are placed through the anterior surface of the muscle 4 to 5 mm. above the cut edge. With scissors the upper half of the tarsus is excised (all but 1.5 mm. of the tarsus may be removed if necessary), and the sutures are passed through the skin; the sutures in the upper part of the muscle are passed through the skin 5 to 6 mm. above the margin of the lid, and the sutures in the lower edge, through the skin at the upper cut edge of the tarsus. If the lower sutures are placed too near the lashes, ectropion will result; if too high, the skin will fall over the lashes and scratch the cornea. The two sets of sutures are tied, and the lower set is cut off: The upper set is used as traction sutures, to pull the upper lid down over the cornea for protection, and anchored with adhesive plaster to the cheek. An ordinary petrolatum dressing is applied, which is replaced with a Fuchs shield in twenty-four hours. All sutures are removed on the sixth day.

All four procedures just described were resections of Müller's muscle. Each surgeon thought that he was excising a part of the

3. Maddox, E. E.: A New Operation for Ptosis, Brit. J. Ophth. **1**:358-362, 1917.

4. Wolff, H.: Die Vorlagerung des Musc. levator palp. sup. (Musc. Mülleri) mit Durchtrennung der Insertion, Arch. f. Augenh. **33**:125-144, 1896.

5. Wolff recognized the difference between the tendon of the levator muscle and Müller's muscle and described two procedures for the correction of ptosis: first, resection of Müller's muscle through the conjunctiva, and, second, resection of the tendon of the levator muscle through the skin.

6. von Blaskovics, L.: A New Operation for Ptosis with Shortening of the Levator and Tarsus, Arch. Ophth. **52**:563-573, 1923; Treatment of Ptosis: Formation of a Fold in the Eyelid and Resection of the Levator and Tarsus, ibid. **1**:672-680, 1929; Eingriffe am Auge, Stuttgart, Ferdinand Enke, 1938, pp. 174-179.

levator muscle. Only Wolff differentiated Müller's muscle from the aponeurosis and from the levator muscle. It is possible that the other operators may have been familiar with the difference between the levator muscle and Müller's muscle, but their failure to differentiate these structures, their descriptions and their illustrations make this doubtful.

In 1942 Agatston [7] modified the Blaskovics procedure, stating that he resected Müller's muscle and the aponeurosis of the levator muscle together. In his procedure the lid is everted and an incision made through the tarsus 3 mm. from its upper margin. The upper part of the tarsus is then dissected upward with scissors, Müller's muscle being cut from the tarsus. The conjunctiva, still attached to the tarsus, is dissected back to the upper fornix. Müller's muscle and the aponeurosis are picked up with forceps, and the tendon is dissected free from the orbicularis muscle up to the areolar tissue of the orbit. Three double-armed sutures are passed from the conjunctival surface through Müller's muscle and the aponeurosis several millimeters above the cut edge, and the two attachments of the levator muscle are cut off 2 to 5 mm. anterior to the sutures. The sutures are then passed anterior to the tarsus and out through the skin, where they are tied over a roll of gauze 2 mm. from the lashes. The part of the tarsus still attached to the conjunctiva is now cut off, and the conjunctiva is approximated to the upper margin of the tarsus with a running silk suture. This is the only operation for ptosis in the literature in which the author claimed to resect both the aponeurosis of the levator muscle and Müller's muscle. Other surgeons may have included both insertions of the levator muscle in their resections, referring to them as the "levator muscle," but their failure to differentiate these structures from the levator muscle itself leads to confusion.

Tucking or Folding of the Aponeurosis of the Levator Muscle.—In 1883 Eversbusch [8] described an operation in which the action of the levator muscle was enhanced by folding the tendon on itself. With the patient under general anesthesia, a Snellen clamp was applied and an incision made through the skin, the orbicularis muscle and the septum orbitale, halfway between the border of the lid and the brow. The upper and the lower lip of the incision were separated to expose the tendon of the levator muscle and the anterior surface of the tarsus. A double-armed suture was passed through the central part of the tendon 3 to 4 mm. from the upper border of the tarsus. Two similar sutures were inserted on each side of the central suture, 3 mm. from it. All three double-armed sutures were then passed between the tarsus and the skin and out through the gray line of the lid. Before they were tied the clamp was removed and the cutaneous incision closed with three other sutures. Tightening the sutures caused the tendon to fold on itself anterior to the tarsus. When a redundancy of skin existed, a strip of skin and of orbicularis muscle was excised, to lessen the weight of the lid and the antagonistic action of the orbicularis muscle.

Snellen [9] suggested two operations for tucking the levator muscle, the first of which was like the Eversbusch procedure and the other did not employ a cutaneous incision. In the latter procedure he passed three sutures from the surface of the skin through the lid just above the tarsus. The needles were then carried up to the fornix, directed downward between the skin and the tendon of the levator muscle and out through the skin 2 mm. from the point of entrance; they were finally tied over beads or pieces of tubing. Wilder [10] and Beard [11] each described an operation for tucking the tendon of the levator muscle and the septum orbitale by means of two sutures placed between the tarsus and the frontalis muscle; they thus combined shortening of the levator muscle with utilization of the frontalis muscle. Except for Eversbusch's operation, none of these tucking procedures attained wide acceptance, and today practically all have been replaced by other methods.

Shortening of the Levator Muscle by Excising a Part of the Lid.—Any operation in which a part of the tarsus is excised results in actual shortening of the levator muscle and should be included here. The ancient Arabian procedure of excising a piece of skin from the upper lid was seldom successful [12] because the skin

7. Agatston, S. A.: Resection of the Levator Palpebrae Muscle by Conjunctival Route for Ptosis: Simplified Technic, Arch. Ophth. **27**:994-996 (May) 1942.

8. Eversbusch, O.: Zur Operation der congenitalen Blepharoptosis, Klin. Monatsbl. f. Augenh. **21**:100-107, 1883.

9. Snellen, H.: New Method of Treating (1) Symblepharon, (2) Ptosis, and (3) Episcleritis, Tr. Ophth. Soc. U. Kingdom **10**:207-210, 1890.

10. Wilder, W. H.: Operation for Ptosis, Tr. Am. Ophth. Soc. **8**:99-102, 1897.

11. Beard, C. H.: Ophthalmic Surgery, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1914, pp. 230-252.

12. Wilder, W. H.: Operations for the Relief of Trichosis, Entropion, Ptosis and Epicanthus, in Wood, C. A.: A System of Ophthalmic Operations, Chicago, Cleveland Press, 1911, vol. 2, pp. 1523-1563.

stretched so as to neutralize the effect of the operation. If enough skin was removed to effect permanent widening of the palpebral fissure, lagophthalmos, corneal ulceration and sometimes loss of the eye resulted. In 1863 von Graefe [13] modified the ancient Arabian procedure slightly by removing an elliptic piece of orbicularis muscle instead of skin from the upper lid. Two years later Bowman,[14] apparently not satisfied with his first attempt to correct ptosis by resection of the levator muscle through the conjunctiva, proposed resection through the skin. He made an incision through the skin, which he reflected upward and downward, then excised an oval piece of orbicularis muscle, tarsus and conjunctiva and sewed the edges together. Gillet de Grandmont [15] accomplished the same thing by placing the cutaneous incision 2 or 3 mm. above the margin of the lid instead of 8 mm. above, as was done by Bowman. Nicati,[16] Boucheron,[17] Gruening,[18] Theobald [19] and Mayou [20] resected a piece of the tarsus. Fergus [21] removed skin, orbicularis muscle and tarsus but spared the conjunctiva. Galezowski [22] removed skin, orbicularis muscle, tarsus and conjunctiva.

Resection of the Tendon of the Levator Muscle Through the Skin.—In 1896 Wolff [4] described a simple method for shortening the tendon of the levator muscle. A cutaneous incision, 2 cm. long, was made at the level of the upper border of the tarsus, and the skin was dissected free from the orbicularis muscle 3 to 4 mm. upward and downward. The orbicularis muscle and the underlying septum orbitale were grasped with forceps and freed from the upper border of the tarsus and the aponeurosis of the levator muscle. Two vertical incisions were made about 10 mm. apart through the aponeurosis of the levator muscle, and two squint hooks were passed beneath the tendon of the muscle. The tendon was then freed from the underlying Müller's muscle; two double-armed surgical sutures were passed through the tendon several millimeters above its attachment to the tarsus and the sutures tied. The tendon anterior to the sutures was excised, the cut edge sutured to the upper border of the tarsus and the skin closed with three or four silk sutures. Wolff recommended 1 mm. of resection for each millimeter of ptosis. De Lapersonne [23] modified the Wolff resection by combining advancement of the insertion with resection of the levator muscle. Instead of attaching the cut edge of the tendon of the levator muscle to the upper margin of the tarsus, he carried the tendon down 2 or 3 mm. from the margin of the lid by passing the sutures through the anterior surface of the tarsus at this point. A piece of skin and orbicularis muscle was sometimes removed before closing the skin. Elschnig [24] resected the tendon of the levator muscle in almost the same way as was outlined by de Lapersonne except that he placed the cutaneous incision 10 mm. above the convex border of the tarsus, rather than 3 to 4 mm. below it. In 1919 Lancaster [25] designed an operation for shortening the levator muscle through the cutaneous route, in which a piece of skin to which the levator muscle was attached was excised, along with the tendon of the levator muscle.

The operations just described are a fairly complete list of the various surgical procedures proposed for increasing the lifting power of the levator muscle. Only a few of these operations are used today, the most popular of which seems to be the Blaskovics and the Wheeler procedure. The Blaskovics technic is the method generally employed for resection of the levator muscle at Stanford University Hospitals,[26] and Greens' Eye Hospital, San Fran-

13. von Graefe, A.: Operation der Ptosis, Arch. f. Ophth. **9** (pt. 2):57-78, 1863.

14. Bowman, W.: Rep. Roy. London Ophth. Hosp. **2**:111-112, 1861.

15. Gillet de Grandmont: Nouvelle opération du ptosis congenital, Bull. et mém. Soc. franç. d'opht. **9**: 80-83, 1891.

16. Nicati, W.: Opération de ptosis par avancement de l'insertion du releveur palpébrale, Arch. d'opht. **10**:162-163, 1890.

17. Boucheron, M.: Opération du ptosis, Arch. d'opht. **8**:229, 1888, cited by von Blaskovics.[6]

18. Gruening, E. C.: The Value of Partial Resection of the Tarsal Cartilage in the Operative Treatment of Congenital Ptosis, Tr. Am. Ophth. Soc. **9**: 574-576, 1902; Treatment of Various Forms of Ptosis by Partial Resection of the Tarsal Cartilage, New York Eye & Ear Infirm. Rep. **11**:1-3, 1904.

19. Theobald, S.: A Simplification of Grandemont's Operation for Ptosis, Tr. Am. Ophth. Soc. **11**: 673-677, 1908.

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21. Fergus, A. F.: Ptosis Operations, Tr. Ophth. Soc. U. Kingdom **28**:184-190, 1908.

22. Galezowski, X., cited by Beard.[11]

23. de Lapersonne, F.: Sur quelques modifications dans les opérations du ptosis, Arch. d'opht. **23**:497-502. 1903.

24. Elschnig, A.: Ptosisoperationen, in Elschnig, A., and Axenfeld, T.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1922, vol. 1, pp. 243-280.

25. Lancaster, W.: The Operative Treatment of Ptosis, Am. J. Ophth. **3**:457, 1920.

26. Bettman, J. W.: Personal communication to the author.

cisco [27]; Passavant Memorial Hospital, Chicago [28]; Illinois Eye and Ear Infirmary, Chicago [29]; University Hospital, Ann Arbor, Mich.[30]; University Hospitals, Minneapolis,[31] and Wills Hospital, Philadelphia.[32] At the Presbyterian Hospital, New York, the Wheeler procedure is the method of choice.[33] At none of these hospitals is Müller's muscle differentiated from the tendon of the levator muscle during the operation.

INDICATIONS FOR RESECTION OF THE LEVATOR MUSCLE

Not all cases of ptosis are suitable for resection of the levator muscle. Most authors [34] agree that some action of this muscle is necessary to insure success of the operation. Blaskovics [6] maintained that his procedure could be used for all types and degrees of ptosis with complete satisfaction, "even when the muscle is absent," and that "an apparent function of the levator develops even in those cases in which this muscle was completely paralyzed or atrophic." It is difficult for me to conceive how an absent muscle can be resected or made to function effectively.

There are two methods of determining the functional state of the levator muscle. First, one can study the action of the upper lid. If the lid is elevated, the palpebral fissure widened or the fold of the upper lid deepened in upward gaze, while the frontalis muscle is kept from acting, there is presumptive evidence that the levator muscle is present and active. Second, according to Koster,[34f] widening of the fissure after the instillation of a few drops of cocaine indicates that Müller's muscle and the levator muscle are present.

ESTIMATION OF THE AMOUNT OF RESECTION FOR 1 MM. OF PTOSIS

Wolff [4] stated that 1 mm. of resection of the levator muscle would correct 1 mm. of ptosis. Kirby [34c] estimated that 1 mm. of ptosis could be corrected by 2 mm. of resection of the levator muscle or by 1 mm. of resection of the tarsus, but added that "it is best to do a little more than this." Green [35] followed the same rule in estimating the amount of shortening necessary. Blaskovics [6] stated that less than 5 mm. of resection has no effect and that no rule can be made for all cases because the effect of all operations for shortening becomes greater with each additional millimeter of resection. In estimating the amount of shortening to be done, one must consider the elasticity of the levator muscle, the weight of the lid, the length of the fissure, the rigidity of the skin, the presence of epicanthus or endophthalmitis and the age of the patient. By pulling on the freed tendon to test its strength and elasticity, Blaskovics estimated the amount of resection necessary. When the levator muscle is elastic as much as 20 mm. may have to be removed for a satisfactory result. When the levator muscle is replaced by scar tissue, more than 10 mm. cannot be removed. If a greater amount must be resected, additional tarsal tissue must be removed. The amount of tissue excised must be measured when the muscle is relaxed, and not when it is stretched by traction.

ADVANTAGES AND DISADVANTAGES OF OPERATIONS ON THE LEVATOR MUSCLE

According to Dunnington,[34b] Malbran [36] and others, shortening of the attachments of the levator muscle in selected cases is the best procedure to use in the repair of ptosis, for the following reasons: (1) Postoperative reaction is negligible; (2) lagophthalmos is almost absent; (3) the lid moves with the globe normally in all directions of gaze; (4) the margin of the lid is not deformed; (5) a good lid fold is produced; (6) the procedure can be used in repair of congenital ptosis even when accompanied with epicanthus; (7) no backward tilt of the head or elevation of the brow results; (8) there is no diplopia after operation due to weakening

27. Green, M. I.: Personal communication to the author.

28. Gifford, S. R.: Personal communication to the author.

29. Gradle, H.: Personal communication to the author.

30. Fralick, F. B.: Personal communication to the author.

31. Burch, F. E.: Personal communication to the author.

32. Spaeth, E. B.: Personal communication to the author.

33. This survey is incomplete and is intended to indicate only a trend. I obtained this information from personal communications.

34. (*a*) Wheeler, J. M.: Personal communication to the author. (*b*) Dunnington, J. H.: Blepharoptosis: Its Surgical Treatment, M. Rec. & Ann. **35**:880-883, 1941. (*c*) Kirby, D. B.: Blepharoptosis: The Technique of Its Surgical Correction, Surg., Gynec. & Obst. **70**:438-449, 1940. (*d*) Spaeth, E. B.: Principles of Ophthalmic Surgery, ed. 1, Philadelphia, Lea & Febiger, 1939. (*e*) Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, 1939. (*f*) Koster, W.: Zur Untersuchung der Muskelverhältnisse des oberen Lides bei Ptosis congenita, Ztschr. f. Augenh. **3**:304, 1900; Die verbounding van den musculus tarsalis sup. bij. ptosis congenita, Nederl. tijdschr. v. geneesk. **2**:417, 1900. Wilder.[12]

35. Green, M. I.: Modified Blaskovics Operation for Ptosis, Bull. Pract. Ophth. **12**:7-9, 1942.

36. Malbran, J.: La corrección operatoria de la ptosis palpebral, Semana méd. **2**:1456-1462, 1941.

of the superior rectus muscle; (9) the method can be used when the superior rectus muscle is weak; (10) and the winking reflex is not disturbed.

The disadvantages of shortening the levator muscle are as follows: 1. The operation can be used successfully only when the levator muscle is present and active. 2. There is a tendency to undercorrection. 3. Lagophthalmos may result if too much tissue is removed.

RESULTS OF SHORTENING THE LEVATOR MUSCLE

In 1923 Blaskovics [6] reported on 21 operations which he had done by his method, with undercorrection in 1 case and overcorrection in 2 cases. In 5 cases there had been total paralysis; in 4 cases, paresis; in 2 cases, an inflammatory condition; in 2 cases, ptosis as a result of trauma; in 4 cases, congenital ptosis and in 4 cases, congenital ptosis with epicanthus. In 1929 he added 12 more cases, in all of which "excellent results" were obtained. Lindner [37] used the Blaskovics operation in 19 cases, the results being excellent in 12 cases, good in 5 cases and unsatisfactory in 1 case, the condition being made worse in 1 case. In 3 cases the operation had to be repeated. Jaensch [38] did this operation in 19 cases, with improvement in all but 1 case. For comparison, the author reviewed the results in 42 cases of ptosis in which resection of the levator muscle was done according to the Wheeler technic. In 30 cases improvement resulted; in 10 cases there was no improvement; and in 2 cases the ptosis was worse. This high percentage of failures was disturbing and led to an examination of the possible causes for such failure.

REASONS FOR FAILURE OF RESECTION OF THE LEVATOR MUSCLE

Among the reasons that resection does not always give a satisfactory correction are the following possibilities: 1. The levator muscle may be too weak to support the lid. In some cases this may be true, especially if no action of this muscle is present. It is obviously impossible to determine before operation the histologic or anatomic condition of the levator muscle. Knowledge of the state of this muscle is therefore entirely dependent on the physiologic behavior of the upper lid. Tests to measure its activity give no information as to the size of the levator muscle, its elasticity, its tensile strength or the presence of fibrosis, degeneration or congenital anomalies of structure or innervation. These factors govern the success of the operation but cannot be determined before operation by physiologic or clinical tests. In acquired ptosis the muscle must be present, even though paralyzed. In cases of congenital ptosis, however, the levator muscle was described as absent or defectively developed by Heuck,[39] Bach,[40] Ahlström [41] and Siemerling.[42] Certainly, if this muscle is absent, an attempt to correct the ptosis by resection of the levator will lead to failure.

2. The sutures may pull out after operation and thus lead to failure. This accident is especially likely to occur if mattress sutures are not employed or if severe edema develops after operation. The danger of the sutures pulling out can be reduced to a minimum by using a double set, as advocated by Blaskovics, and by leaving a generous amount of the tendon anterior to the sutures. Postoperative edema can be minimized by avoiding unnecessary trauma during the operation.

3. An insufficient amount of the levator muscle may be resected in some cases, especially if the muscle is extremely elastic, the lid thick and heavy or the surgeon timid. Blaskovics called attention to the fact that the amount of resection needed must be determined at the time of operation by the condition of the levator muscle and the lid, rather than by a rule calling for so many millimeters of resection for so many millimeters of ptosis.

4. Resection of Müller's muscle instead of the tendon of the levator muscle may be responsible in some cases. Unfortunately, most textbooks on ophthalmic surgery do not differentiate these two structures or call attention to the possibility of this mistake being made during the operation. Even the sponsors of some operations for resection of the levator muscle have failed in this respect.

A brief study of the anatomic position of Müller's muscle will convince any one that every resection of the levator muscle done through

37. Lindner, K.: Ueber die Ptosisoperation nach Blaskovics, Klin. Monatsbl. f. Augenh. **93**:1-12, 1934.

38. Jaensch, P. A.: Zur Ptosisoperation, Klin. Monatsbl. f. Augenh. **94**:183-189, 1935.

39. Heuck, A.: Ueber angeborenen vererbten Beweglichkeits-Defekt der Augen: Fall IV, Klin. Monatsbl. f. Augenh. **17**:253-278, 1879.

40. Bach, L.: Anatomischer Befund eines doppelseitigen angeborenen Kryptophthalmos beim Kaniñchen nebst Bemerkungen über das Okulomotoriuskerngebeit. Arch. f. Augenh. **32**:16-32, 1892.

41. Ahlström, G.: Doppelseitige kongenitale Ptosis und Unbeweglichkeit der Bulbi, Beitr. z. Augenh. **2**: 523-554, 1895.

42. Siemerling, E.: Anatomischer Befund bei einseiteger congenitaler Ptosis, Arch. f. Psychiat. **23**:764-774, 1892.

the conjunctiva is, at least in part, a resection of Müller's muscle, for Müller's muscle lies between the conjunctiva and the tendon of the levator muscle. Few surgeons have recognized or called attention to this fact. Bowman, who performed the first resection of the levator muscle on record, "everted the lid and excised the posterior or upper edge of the palpebral cartilage, with about half an inch of the tendon of the levator palpebrae inserted into it." According to this description, he resected Müller's muscle. He may have included with Müller's muscle a piece of the aponeurosis, but his failure to differentiate these structures makes it doubtful. Blaskovics,[6] after everting the lid and reflecting the conjunctiva, grasped the exposed "levator muscle" and freed it from the tarsus below, from the conjunctiva behind and from the "pretarsal" tissues in front. The muscle, which he called the "levator muscle," must have been Müller's muscle. It is conceivable that he may have pulled the levator muscle into the wound by strong downward traction and thus have resected a part of it. Or he may have placed his sutures deep enough in Müller's muscle to have included the aponeurosis and thus have resected it also. His failure to differentiate these structures is unfortunate. Maddox[8] expressed vividly his conception of the levator muscle by saying:

> After double eversion of the eyelid, nothing intervenes between the surgeon and the levator tendon but a thin loose layer of that peculiarly accommodating membrane, the conjunctiva, which is no sooner divided along the upper margin of the tarsus and reflected from the tendon, than the latter lies in full view, and can be shortened without interfering at all with the natural beauty of the front of the eyelid.

Obviously he was describing Müller's muscle.

The tissues of the upper lid above the tarsus from the inner surface out are (1) the conjunctiva, (2) Müller's muscle, (3) the aponeurosis of the levator muscle, (4) the septum orbitale, (5) the orbicularis muscle and (6) the skin (fig. 1). It is therefore evident that the first structure encountered by the surgeon after the conjunctiva has been reflected is Müller's muscle, and not the levator muscle, as is so often stated by authors of operations for resection of the levator muscle. The levator muscle ends 10 or 12 mm. from the tarsus and cannot be reached through the conjunctiva unless Müller's muscle and the aponeurosis are strongly pulled downward. What Bowman, Wolff, Blaskovics, Maddox and others described and illustrated as the levator was Müller's muscle. The question therefore arises: Did these surgeons resect only Müller's muscle, or did they, unknowingly, resect also the aponeurosis of the levator muscle, which lies anterior to and loosely connected with Müller's muscle?

Because Müller's muscle has been so often confused with the levator muscle and with its tendon (aponeurosis), it is entirely possible that only Müller's muscle may have been resected in some cases. Considering the frail and delicate character of this muscle, it is probable, as suggested by Hildreth,[43] that this fact alone may have been responsible for some failures following supposed resection of the levator muscle.

The problem was approached in the following manner: First, the normal anatomy of the upper lid and the levator muscle was studied by dissection of orbits of cadavers in the usual three dimensional manner—from the anterior, the lateral and the superior aspect. This investigation was supplemented by studies of gross sections of other cadaver orbits in the frontal, the sagittal and the horizontal plane. Second, an attempt was made to demonstrate the normal planes of cleavage between (*a*) the conjunctiva and Müller's muscle, (*b*) Müller's muscle and the tendon of the levator muscle and (*c*) the tendon and the septum orbitale. Different-colored material was placed between these three layers; the tissues were replaced in their normal positions, and the orbit was sectioned in the sagittal plane for study. Third, three types of operations for resection of the levator muscle

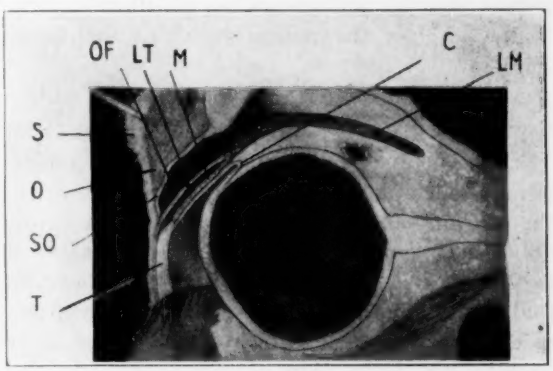

Fig. 1.—Sagittal section through the orbit, showing the normal anatomic structure of the upper lid. The tissues have been separated to show the normal relation of the conjunctiva (*C*), Müller's muscle (*M*), the tendon of the levator muscle (*L T*), the septum orbitale (*S O*), the orbicularis muscle (*O*) and the skin (*S*). The section also shows the position of the tarsus (*T*), the orbital fat, removed (*OF*), and the levataor muscle (*L M*) (retouched photograph of a specimen from a cadaver).

43. Hildreth, H. R.: The Insertions of the Levator Palpebrae Muscle, Am. J. Ophth. **24**:749-758, 1941.

were done on cadavers in the routine manner, and the orbits were sectioned in the sagittal plane and then studied to determine what tissue or tissues had been removed.

ANATOMY OF THE LEVATOR MUSCLE AND THE UPPER LID

With the texbooks of Whitnall,[44] Wolff [45] and others [46] as a guide, a detailed study of the normal anatomy of the upper lid and the levator muscle showed that the skin, the orbicularis muscle and the septum orbitale hang loosely from the superior orbital margin to the tarsus. The levator muscle arises at the apex of the orbit and runs forward in close contact with the underlying superior rectus muscle to above the globe, where the two become separated by a thickening of Tenon's capsule. At this point the aponeurosis and Müller's muscle begin and extend forward and downward for 10 to 12 mm., to the level of the upper border of the tarsus (fig. 1).

More detailed study of these parts with a magnifying glass brings out the following facts:

1. The skin is firmly adherent to the underlying tissues at the brow and extends down to the margin of the lid, where it is again firmly adherent. Five to 10 mm. above the lashes it is thrown into a horizontal fold, the upper lid fold, by the terminal fibers of the aponeurosis of the levator muscle, which end here and serve to keep the loose skin of the upper lid from prolapsing over the lashes (fig. 1). Elsewhere the skin is loosely attached to the underlying orbicularis muscle except at the internal and external canthal ligaments, to which it is firmly attached.

2. The orbicularis muscle is fused with the corrugator supercilii muscle and the frontalis muscle at the brow (fig. 1). From here it extends downward to the margin of the lid, being loosely attached to the skin in front and to the septum orbitale behind. Anterior to the tarsus the orbicularis muscle is loosely adherent to the skin in front and to the tarsus behind; medially it is attached to the nasal margin of the orbit and the internal canthal ligament and laterally to the bone of the temporal margin of the orbit by way of the external raphe. At the level of the upper lid fold it is fused with some of the terminal fibers of the aponeurosis of the levator muscle as they pass through the orbicularis muscle to become attached to the skin.

3. The septum orbitale begins at the orbital margin from the junction of the periosteum of the frontal bone and the periorbita of the orbit (fig. 1). At this point it is thickest, and from here downward it becomes thinner, as it hugs the posterior surface of the orbicularis muscle. As a matter of fact, it seems more accurate surgically to conceive of this structure as the posterior fascial sheath of the orbicularis muscle rather than as a separate structure. At about the level of the upper border of the tarsus, the septum orbitale seems to end by fusing with the anterior surface of the aponeurosis of the levator muscle, thus forming a barrier to the prolapse of the orbital fat. Below the upper border of the tarsus and between the anterior surface of the tarsus and the posterior surface of the orbicularis, there is a thin layer of loose connective tissue, which occupies the same relation to the orbicularis muscle as does the septum orbitale above and which for practical purposes can be considered a prolongation of this membrane. Medially the septum orbitale is adherent to the nasal margin of the orbit and laterally to the temporal margin.

4. Posterior to the septum orbitale above the tarsus is the orbital fat (fig. 1), which is comma-like in sagittal section, being thick and rounded in front and thin and pointed behind. It fills the interval between the sheath of the levator muscle, below; the roof of the orbit, above, and the septum orbitale, in front.

5. Below and behind this roll of orbital fat is the aponeurosis of the levator muscle, with a thin, transparent sheath of areolar tissue (fig. 1), which blends with the septum orbitale. The aponeurosis arises from the anterior extremity of the terminal fibers of the levator muscle and extends forward and downward to the tissues of the upper lid anterior to the tarsus (fig. 1). It is divided into a medial horn and a lateral horn. The former is thin and fragile and is difficult to follow in its course from the levator muscle to the upper border of the medial canthal ligament. In this position it is posterior to the septum orbitale and anterior to Müller's muscle. The lateral horn is more dense and fibrous; it extends from the lateral half of the

44. Whitnall, S. E.: Anatomy of the Human Orbit and Accessory Organs of Vision, ed. 2, London, Oxford University Press, 1932, pp. 115-192.

45. Wolff, E.: Anatomy of the Eye and Orbit, ed. 2, Philadelphia, The Blakiston Company, 1940, pp. 120-139.

46. Eisler, P.: Die Anatomie des menschlichen Auges, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, pp. 211-386. Wolff, H.: Ueber die Sehne des Musculus levator palpebral superioris, Ztschr. f. Augenh. **13**:440, 1905; **15**:576, 1906.

levator muscle to the upper border of the external canthal ligament, posterior to the septum orbitale and anterior to Müller's muscle. It separates the lacrimal gland into two parts as it proceeds along its course, so that the main part of the gland lies above the lateral horn and the accessory lobe is behind it.

The middle part of the tendon of the levator muscle is the portion most concerned with operations for ptosis. It is about 10 to 12 mm. long and extends from the anterior extremity of the levator muscle forward, between the orbital fat, above, and Müller's muscle, below, to the upper border of the tarsus. Here it divides into thousands of fine fibrillae, which become attached in small part to the anterior surface of the tarsus but chiefly to the overlying orbicularis muscle and the skin, thus producing the upper lid fold. The septum orbitale fuses with the anterior surface of the tendon of the levator muscle a short distance above the tarsus (fig. 1).

6. Müller's muscle is about 10 or 12 mm. long and extends from the levator muscle to the upper border of the tarsus (fig. 1). At its origin from the levator muscle, 2 to 3 mm. posterior to the origin of the aponeurosis, there is a gradual transition from striated to smooth muscle fibers. The muscle is about 0.5 mm. thick and is delicate and easily torn. It is loosely adherent above to the aponeurosis and is adherent below to the conjunctiva.

7. The conjunctiva is firmly adherent to the tarsus below and is loosely attached to Müller's muscle above (fig. 1). At the upper fornix it is supported by a thin sheath of connective tissue, extending forward from the conjoined sheaths of the levator and the superior rectus muscle, i. e., by the so-called suspensory ligament of the upper fornix.

With careful gross dissection of the upper lid, in sagittal sections especially, one observes three layers of tissue below the upper border of the tarsus, namely, the skin, the orbicularis muscle and the tarsus, with the adherent conjunctiva (fig. 1). These layers can be easily separated surgically. Above the upper border of the tarsus, the orbital fat divides the tissues of the upper lid into two layers, so that anterior to the fat are the skin, the orbicularis muscle and the septum orbitale. Posteriorly there are the aponeurosis of the levator muscle, Müller's muscle and the conjunctiva. These layers can be easily identified and separated in the cadaver (fig. 1).

For further study and identification of these planes of dissection, the following procedure was used to simulate as much as possible the conditions encountered surgically: The upper lid was everted and the conjunctiva dissected free from Müller's muscle for 10 to 15 mm. Then the upper border of the tarsus was incised, and the attachments of the levator muscle were dissected upward along the pretarsal fascial plane for a distance of 10 to 15 mm.[47] Two vertical incisions, about 15 to 20 mm. apart, were then made through the pretarsal fascial tissues, and a tunnel was formed between these two incisions by blunt dissection with scissors. A clamp was applied to the tissue thus raised, and the latter was freed from its lower attachment and

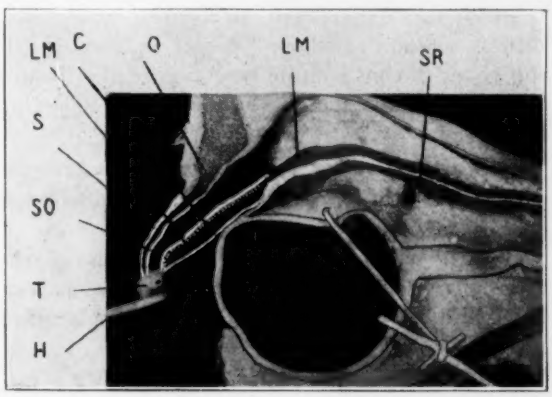

Fig. 2.—Sagittal section through the orbit, showing that the tendon of the levator muscle and Müller's muscle (*L M*) are separated as one layer of tissue in the routine dissection involved in resection of the levator muscle (*L M*) through the conjunctiva (*C*). *H* indicates the hook; *T*, the tarsus; *S O*, the septum orbitale; *S*, the skin; *O*, the orbicularis muscle, and *S R*, the superior rectus muscle (retouched photograph of a cadaver specimen).

dissected upward for 10 to 12 mm.[48] Three layers of tissue were thus separated—the conjunctiva, Müller's muscle and the pretarsal fascia. Pieces of colored celluloid were then inserted between these layers, so that a blue piece was placed between the conjunctiva and Müller's muscle, a red piece, between Müller's muscle and the pretarsal tissue and a green piece, between the pretarsal tissue and the skin. The entire orbit was then sectioned in the sagittal plane, and the fascial planes and the tissue layers

47. In this dissection it was certain that Müller's muscle, which is always attached to the upper border of the tarsus, was dissected upward. But at the time it was not definitely determined whether the aponeurosis was also included.

48. The tissue plane of this dissection seemed to be entirely in the plane of the orbicularis muscle.

were studied. From behind forward, the first layer of tissue was the conjunctiva, to which a few fibers of Müller's muscle were still attached (fig. 2). The first plane of dissection, marked by blue celluloid, passed between Müller's muscle and the conjunctiva. The second layer of tissue, to which the tarsus was attached, was made up of Müller's muscle, behind, and the levator aponeurosis, in front. The second plane of dissection, marked by red celluloid, passed between the aponeurosis, behind, and the septum orbitale, in front. The third layer of tissue was made up of septum orbitale and orbicularis muscle. The third plane of dissection, marked by green celluloid, passed through the orbicularis muscle, with the skin in front and the septum orbitale behind.

From this experiment, performed under conditions simulating those present in the operating room during routine resection of the levator muscle through the conjunctiva, the following conclusions were drawn:

1. A natural plane of cleavage exists between Müller's muscle and the conjunctiva, making it easy to separate the two.

2. Müller's muscle and the tendon of the levator muscle are removed together as one layer and are not easily separated or identified under these conditions.

3. The "pretarsal" fascia, anterior to the tarsus, is the same as the posterior fascial sheath of the orbicularis muscle and becomes continuous with the septum orbitale above the tarsus.

EXPERIMENTAL RESECTIONS OF THE LEVATOR MUSCLE

To test these conclusions further under conditions simulating those present in the operating room, three types of operation for resection of the levator muscle were done on cadavers, and the orbits were later sectioned sagitally, to determine how much and what tissue was removed.

1. *Resection of the Levator Muscle According to the Wheeler Technic.*—The right orbit of a cadaver was subjected to resection of the levator muscle according to the technic practiced by Wheeler. The lid was everted by means of an Ehrhardt clamp. The conjunctiva was incised 1 to 2 mm. above the upper border of the tarsus and dissected free from Müller's muscle for 10 to 12 mm. An incision was then made through the tarsus 1 to 2 mm. from its upper border down to the pretarsal fascia. The excised tarsus was grasped with forceps, and the tissues attached were dissected upward from the orbicularis muscle for 10 to 12 mm. Three double-armed silk sutures were passed through the conjunctiva near its cut edge and then through the attachments of the levator muscle 10 mm. from the excised tarsus. The excised tissue was only 5 mm. long in its relaxed state. The sutures were then passed through the cut edge of the tarsus and through the skin and tied over a rubber peg. The excised tissue was sent to the laboratory for histologic study and found to consist of tarsus, Müller's muscle and a connective tissue layer representing the tendon of the levator muscle. The orbit was then sectioned in the sagittal plane, and the tissues were studied grossly. This showed that less than one half of Müller's muscle and the tendon of the levator muscle had been removed (fig. 3).

This procedure closely approximated that practiced routinely by the late Dr. Wheeler for

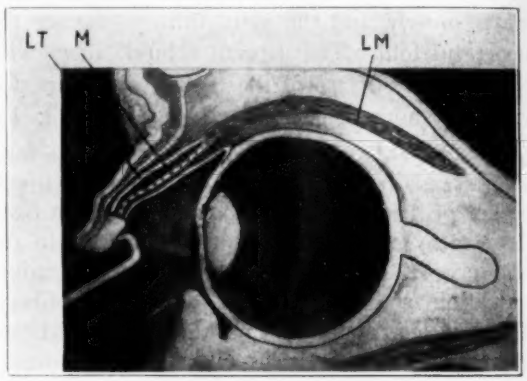

Fig. 3.—Sagittal section through the orbit after routine resection of 10 mm. of tissue of the levator muscle (*L M*) according to the Bowman-Wheeler technic. Note that only a small part of the tendon of the levator muscle (*L T*) and Müller's muscle (*M*) has been removed (retouched photograph of a cadaver specimen).

mild ptosis. It was obvious, from the small amount of tissue removed, about 5 mm., that undercorrection would result in the patient, having severe ptosis. In justice to the operation, it was decided to repeat the procedure on another cadaver, to determine whether this technic could be modified by simply removing more tissue of the levator muscle and thus made useful for severe ptosis. Therefore resection of the levator muscle was done on another cadaver, according to the preceding technic except that instead of resecting what appeared to be 10 mm. of the levator tissue, as was done the first time, an apparent 20 to 25 mm. was removed. The excised tissue when relaxed measured only 18 mm. in length [49] and was sent to the laboratory

49. It was difficult to measure the excised tissue accurately because of its elasticity and lack of tone. If the tissue was stretched horizontally, it became shorter vertically, and vice versa.

for histologic study. The specimen consisted of a small part of the tarsus, a layer of Müller's muscle, most of the tendon of the levator muscle and a few striated muscle fibers from the levator muscle. The orbit was then sectioned in the sagittal plane, and the tissues were studied grossly (fig. 4). Practically all of Müller's muscle and the aponeurosis of the levator

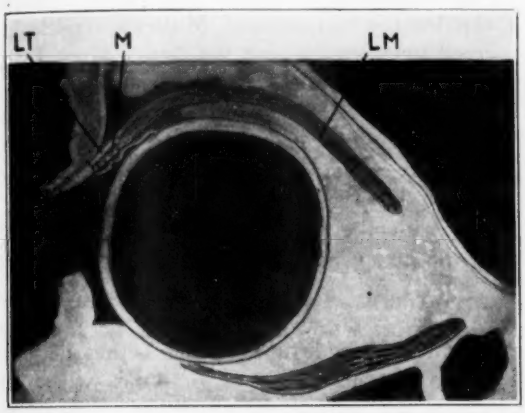

Fig. 4.—Sagittal section through the orbit after resection of a large amount of the levator muscle (*L M*) according to the Bowman-Wheeler technic. Note that all but 2 or 3 mm. of the tendon of the levator muscle (*L T*) and Müller's muscle (*M*) has been removed (retouched photograph from cadaver specimen).

muscle had been removed. The tissues excised consisted, therefore, of both Müller's muscle and the tendon of the levator muscle.

From this study it may be concluded that resection of Müller's muscle through the conjunctiva, according to the procedure just outlined, must include the tendon of the levator muscle as well as Müller's muscle, and that a natural plane of cleavage exists between Müller's muscle and the conjunctiva, on the one hand, and between the aponeurosis and the septum orbitale, on the other. It also demonstrates that one can, with effort, resect almost all the attachments of the levator muscle with this technic. Traction on the tissues of the levator muscle stretches them and gives an erroneous impression of how much tissue is being removed.

2. *Resection of the Levator Muscle According to the Blaskovics Technic.*—A third cadaver was subjected to the Blaskovics procedure. The upper lid was everted by a Desmarres lid evertor, and an incision was made through the conjunctiva 3 to 4 mm. from the upper border of the tarsus. The conjunctiva was freed from Müller's muscle, and three sutures were passed through the cut edge, to be used for traction. Three other sutures were then passed through Müller's muscle 3 to 4 mm. from the tarsus and used for traction. The tissues attached to the upper border of the tarsus were cut across below the sutures and dissected free from the conjunctiva, on one side, and the pretarsal fascia, on the other, for 20 to 25 mm. During this procedure the lid evertor was removed and the upper lid retracted upward to give a better exposure. The upper half of the tarsus was now excised, and the three double-armed sutures in the conjunctiva were passed through the levator muscle 20 to 25 mm. from its cut edge and left loose. Another set of double-armed silk sutures was then passed through the anterior surface of the levator muscle 5 to 6 mm. above the line of the other sutures. The upper row of sutures was passed through the skin 5 to 6 mm. from the border of the lid and left loose. The lower set of sutures was then passed between the tarsus and the orbicularis muscle and brought out through the skin 2 mm. from the lashes. The upper set of sutures was tied over a rubber peg and cut off, while the lower set was tied without the use of a rubber peg. The excised tissue was sent to the laboratory for histologic study. The specimen consisted of Müller's muscle, the tendon of the levator muscle and a large part of the anterior end of the striated muscle fibers of the levator muscle. The orbit was then sectioned in the sagittal plane, and the tissues were

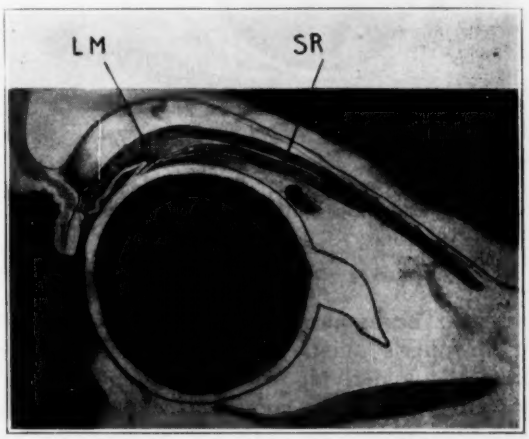

Fig. 5.—Sagittal section through the orbit after resection of a large amount of the levator muscle according to the Blaskovics technic. Note that all of the tendon of the levator muscle and all of Müller's muscle have been removed (retouched photograph of a cadaver specimen).

studied grossly. Inspection showed that all of Müller's muscle and the aponeurosis had been excised and that the sutures had been placed in the anterior end of the striated muscle fibers of the levator muscle (fig. 5).

This experiment shows that with the Blaskovics technic for resection of the levator muscle both Müller's muscle and the aponeurosis, as

well as part of the levator muscle itself, were resected.

3. *Resection of the Levator Muscle Through the Skin.*—The left orbit of a fourth cadaver was subjected to resection of the levator muscle through the skin. A lid clamp was inserted far up into the upper fornix and an incision made through the skin 10 mm. above the lashes. The skin was reflected upward and downward 4 to 5 mm. and an incision made through the orbicularis muscle and the septum orbitale, exposing the orbital fat and the anterior surface of the aponeurosis of the levator muscle. A retaining speculum was inserted to hold the upper lip of the wound and the fat out of the way and to expose the attachment of the aponeurosis to the tarsus. The aponeurosis of the levator muscle and Müller's muscle were then dissected free from the underlying conjunctiva and reflected upward for a distance of 10 to 12 mm. A clamp was then placed on both the aponeurosis and Müller's muscle, and, while traction was exerted downward, the levator muscle was dissected free from the orbital fat, above, and the conjunctiva and Tenon's capsule, below. Three double-armed sutures were passed through the levator muscle from behind forward 15 to 20 mm. from its lower edge, and the tissue anterior to the sutures was excised. The sutures were then passed through the upper border of the tarsus and out through the skin 8 to 10 mm. above the lashes and tied over a rubber peg. The orbit was then sectioned in the sagittal plane, and the tissues were studied grossly. They showed that all of Müller's muscle and the aponeurosis of the levator muscle had been excised and that the sutures had been passed through the anterior end of the levator muscle (fig. 6).

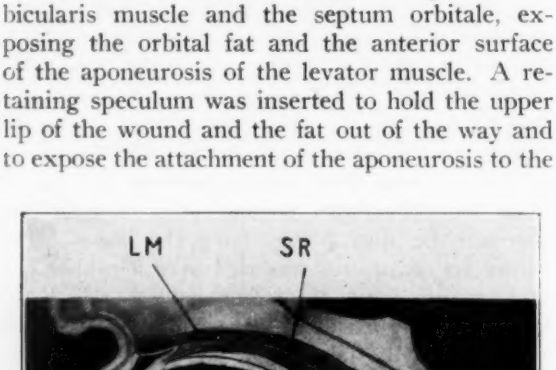

Fig. 6.—Sagittal section through the orbit after resection of a large amount of the levator muscle (*L M*) done by the cutaneous route. Note that all of the tendon of the levator muscle and all of Müller's muscle have been removed. *S R* indicates the superior rectus muscle (retouched photograph of a cadaver specimen).

This experiment revealed that the aponeurosis of the levator muscle and Müller's muscle are resected together through the skin. Much better exposure is obtained when resection of the levator muscle is performed through the skin. Also, more muscle can be excised, the sutures more firmly anchored and more accurate closure effected. It is impossible to confuse Müller's muscle with the tendon of the levator muscle when the levator muscle is resected through the skin.

SUMMARY

Resection of the levator muscle is the operation of choice for the correction of ptosis when this muscle is not completely paralyzed. There are various surgical procedures for increasing the lifting power of the muscle. Inadequate correction of ptosis following resection of the levator muscle through the conjunctiva may be due to unintentional resection of Müller's muscle, instead of the tendon of the levator muscle. Anatomic studies of the upper lid and of the levator muscle in cadavers demonstrated that Müller's muscle is never resected alone but that the tendon of the levator muscle and Müller's muscle are always excised together as one layer in the routine resection of the levator muscle.

CONCLUSIONS

Under conditions simulating those in the operating room, Müller's muscle and the tendon of the levator muscle are always excised together in the routine resection of the levator muscle as done through the conjunctiva.

When resection of the levator muscle gives inadequate correction of ptosis, failure may be due to resection of Müller's muscle instead of the tendon of the levator muscle.

Inadequate correction of ptosis following resection of the levator muscle may be due to insufficient resection, pulling out of the suture or absence of the levator muscle.

Prof. S. R. Detwiler, director of anatomy, Columbia University College of Physicians and Surgeons supplied the cadavers used in this study.

430 Union Street.

UVEITIS, WITH POLIOSIS, VITILIGO, ALOPECIA AND DYSACOUSIA

(VOGT-KOYANAGI SYNDROME)

CAPTAIN EMANUEL ROSEN

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Bilateral uveitis associated with alopecia, poliosis, vitiligo and dysacousia has been described in ophthalmic literature 47 times. Recently the name Vogt-Koyanagi syndrome has been offered to supplant these harsh sounding terms,[1] and Martínez [2] has attempted to rename the syndrome Vogt-Harada disease. The latter suggestion has a further purpose than mere lingual torsion, for Martínez has tried to link the signs and symptoms of the more malignant Vogt-Koyanagi syndrome with those of the usually benign Harada disease. According to Babel's definition a case of nontraumatic bilateral uveitis accompanied by any or all of the associated signs—namely, poliosis, vitiligo, alopecia and dysacousia—should be regarded as an instance of the Vogt-Koyanagi syndrome.[3] This definition has been accepted for the purposes of the present study.

It is known that the disease in question has a protracted course, responds poorly to treatment and usually terminates in partial or complete blindness. The purpose of this paper is to present a case of the disease, to draw some conclusions about its nature and course and to suggest a method of treatment.

REPORT OF A CASE

The patient, a Puerto Rican residing in New York, reported to the ophthalmologic clinic at the station hospital on Feb. 25, 1943, complaining of blurred vision in both eyes. He desired a change in his glasses, which were six months old and which seemed never to have improved his vision much. The patient was 24 years old and had been in the United States for four years. He had been in the service only five months and believed his vision had gradually become worse during that time.

History.—The patient's family history showed no record of any pigmentary, ocular or cutaneous disease. There was no family history of consanguinity. The patient's parents were both alive and well. The father, who was 60 years old, had had graying of the hair during the last four years. There were four brothers and three sisters. No member of the family had shown any evidence of alopecia, poliosis, dysacousia or vitiligo. There was no family history of tuberculosis, syphilis, diabetes, nephritis or mental or nervous disease. The patient did not recall any sudden vasomotor, neurotic, pigmentary or depigmentary phenomena in members of the family.

There had been no unusual circumstances attending the patient's birth. He said he had not had any childhood diseases other than chickenpox, measles and scarlet fever, these conditions having been rather mild and having produced no serious complications.

The patient had no recollection of having had malaria, typhoid or any "parasites." He had never had an abnormal reaction to a drug, serum or foreign protein. There had been no personal history of asthma, hay fever or other allergic manifestations, though two of his sisters had had asthma. He said also that he had not had any venereal disease; he gave no history of any generalized eruption or disturbance of the mucous membrane.

The patient had never been hospitalized for either a surgical or a medical ailment until the onset of the present disease of the eyes. In 1941, while riding a bicycle, he had become involved in an automobile accident, in which his left forearm and wrist were injured. There was no history of unconsciousness, the patient having been retained for only a few hours at the hospital.

The patient's difficulties with his eyes began in 1938, when he became aware of difficulty in reading newspapers and books. There was no difficulty for distant vision. There was no acute sudden onset, and there existed at no time any of the acute symptoms which usher in an acute febrile disease. At the time of onset of these ocular symptoms the patient was in Puerto Rico and was swimming under water a great deal. He believed this activity caused his eyes to become severely inflamed and to remain so for the greater part of the day. He consulted his family physician regarding this redness of his eyes. "Eye drops" and some form of internal medication were prescribed, and the patient was advised to continue this therapy for one month. At the end of this time no improvement was noticed, and the patient sought aid in San Juan, Puerto Rico, finally securing the services of an ophthalmologist, whose diagnosis was bilateral uveitis. Treatment was given accordingly, the patient making weekly visits to this physician for a total of six visits. During this period the patient was given a solution of drops to instil into his eyes three times daily, internal medication and hot compresses. No focal investigation was made further than to uncover and have removed seven infected teeth. A Wassermann test of the blood was reported as giving a negative reaction. On the fourth day after the original attack the patient noticed the appearance of white eye-

1. Babel, J., cited by Carrasquillo, H. F.: Uveitis with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **28**:385 (Sept.) 1942.

2. Martínez, L., Jr.: An. Soc. mex. de oftal. y oto-rino-laring. **16**:273, 1941.

3. Carrasquillo, H. F.: Uveitis with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **28**:385 (Sept.) 1942.

lashes. Two white patches of hair, symmetrically located in the occipital area, appeared about this same time. The vision rapidly diminished and was considerably reduced for distance perception. The patient observed that if he directed his gaze toward the ceiling he was able to see the floor more clearly.

After six weeks of treatment at San Juan vision was much improved, and the patient of his own accord discontinued medical care. One year later there occurred a second attack, which was much more pronounced in the right than in the left eye. No medical aid was sought on this occasion. After several weeks his eyes again began to clear up, though not completely. In the spring of 1942 the patient again noticed a gradual

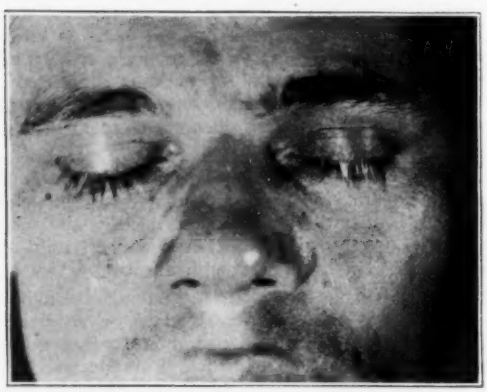

Fig. 1.—Photograph of the patient's eyelids and brows, showing several white eyelashes.

blurring of vision, which became progressive and appeared much more pronounced in the left eye. This left eye became much worse during succeeding summer months. In the autumn of 1942 the patient was seen at one of the clinics of the station hospital. His glasses were changed, but there was no great improvement in his ability to read.

In none of the four attacks which the patient described was the onset accompanied or introduced by any acute symptoms, such as nausea, vomiting, headache or fever. At no time had there been pain of any sort—dizziness, tinnitus, deafness, lassitude, anorexia, stiffness of the neck or febrile reaction.

Examination.—The patient was a short, dark Puerto Rican, 5 feet 1 inch (155 cm.) in height, weighing 128 pounds (58 Kg.) and fairly well built. He had thick hair, which was dark except in the region of the two occipital prominences; here there were some small symmetric patches of white hair. There were some small white eyelashes on each of the lids (fig. 1). Examination of the axillary and pubic regions failed to show any white hairs. Sparseness of hair was noted on the temporal portion of each eyebrow.

There was a mild papular eruption on the patient's forehead and on both malar regions. Careful examination of the skin of the back, the shoulders, the chest and the arms showed several small depigmented islands, none of which was more than 10 mm. in its greatest diameter; these islands were symmetric both in location and in appearance. The vitiligo was present on the shoulders and the chest and was not visible elsewhere.

The results of otolaryngologic consultations were reported as follows: The nose was essentially normal; the pharynx showed infected tonsils; the larynx was normal, and all the sinuses transilluminated readily. The wall of the left auditory canal and the left tympanic membrane were normal, whereas the right eardrum showed a scar on the membrane at 7 o'clock. Weber and Rinne tests gave negative results. An audiogram was taken and found normal.

Examination of the teeth showed that several upper teeth were lacking. All the remaining teeth were in good condition, with no indication of abscess or of involvement of the gums. There was some slight recession of the gums.

No abnormality was noticed as regards pigmentation of the breast. The heart and the lungs were not remarkable.

There were several white eyelashes in both the upper and lower lids. A faint bilateral ciliary injection was present in each eye. The pupils were equal but semidilated (5 mm., with a retarded and minimal reaction both to light and in accommodation but with some response). No abnormalities of muscle were noted. There was nothing remarkable about the conjunctival surface of the lids or about the lacrimal apparatus. The anterior chambers were rather deep. Keratic precipitates were visible on the corneal surfaces with the ordinary loupe (fig. 2). Some pigment was also present on the anterior capsule of each lens and became much more readily visible when the pupil was dilated. The slightly different color of the inner circle of the iris was striking. Many minute "fluffs" of exudate were seen encircling the pupillary margin, probably a

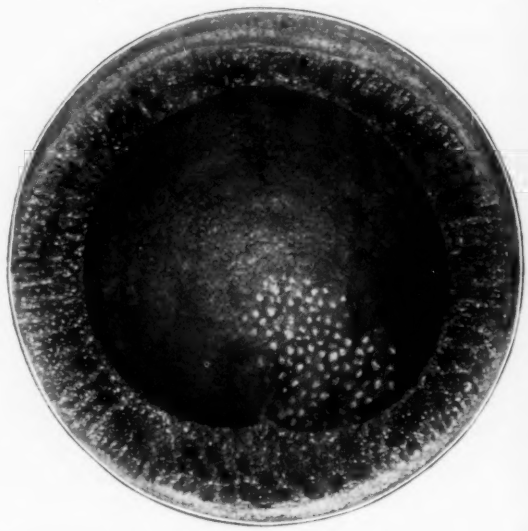

Fig. 2.—Drawing of the iris and the pupil of the patient's right eye.

dozen of these flocculi being present within each pupillary circumference. Between the flocculi was a thin area of exudate plastered on the capsule of the lens.

One small posterior synechia was present in the right eye at 6 o'clock. The vitreous appeared clear except for an occasional minute vitreous "floater."

Slit lamp studies of the cornea showed deposition of precipitates bilaterally. The number was much greater in the left eye. The keratic precipitates were not deposited in the triangular manner usually described for this condition but were for the most part crowded around the summit of the cornea. The precipitates were made up of various-sized islands which en masse pro-

duced a disklike arrangement. The larger precipitates, which were pellucid and crenelated, were definitely nonpigmented but had a lardaceous consistency. The smaller deposits between the larger ones had slight pseudopods resembling melting ice. In the anterior chamber some fine dustlike grains could be seen moving slowly. The aqueous flare was pronounced. There was no real atrophy of the main structure of the iris, for diaphanoscopy did not allow the passage of light rays except at the pupillary border. The entire pupillary border of each eye had a moth-eaten appearance. None of the small accordion-like folds of this portion of the iris were visible. The "chewed-out" effect made the inner circle of the iris look much like a gray border in a brown iris. In the right eye at 6 o'clock there was a peculiar synechia which folded down behind the iris to be attached to the capsule of the lens like a tree trunk with two large limbs growing out of the ground. The structure of the iris itself was not abundantly convoluted and suggested a primitive type of iris. The small flocculi seen with the ordinary loupe looked much more fluffy under the slit lamp, for they were translucent and resembled cotton balls. Some deposition of the pigment of the iris could be seen in these "fluffs," as well as in the exudate between them. There were also many minute granules of pigment on the anterior capsule of the lens, along with some rather solid-looking remnants of dense brown pigment. The lens appeared to be clear throughout, but when the focus was carefully directed on the part of the posterior capsule of the lens in the zone of specular reflection a polychromatic luster was obtained. The reflex from the vitreous was not easily obtained, since the corneal precipitates were clustered closely about the central corneal area. When a narrow beam of light was played well into the anterior third of the vitreous, difficulty was encountered in bringing out the framework of the vitreous. There were two reasons for this difficulty: First, there was no absorption of rays of light by the pigment epithelium of the retina, so that the effect was like that seen in an albino; secondly, the framework of the vitreous was decomposed. The vitreous resembled that of a high myope, wherein the strands of the framework are brown and bare and the horizontal interfibrillar substance has disappeared. Within the fluid clefts of the vitreous some minute brownish granules could be seen. In the right eye just a short distance behind the retrolental area was a definite fluid cleft resembling a small detachment of the vitreous. The generalized return "glow" of the illuminating beam masked a good many characteristics of the vitreous, but the thin, rootlike appearance seemed to be the predominant characteristic of the degeneration of the vitreous.

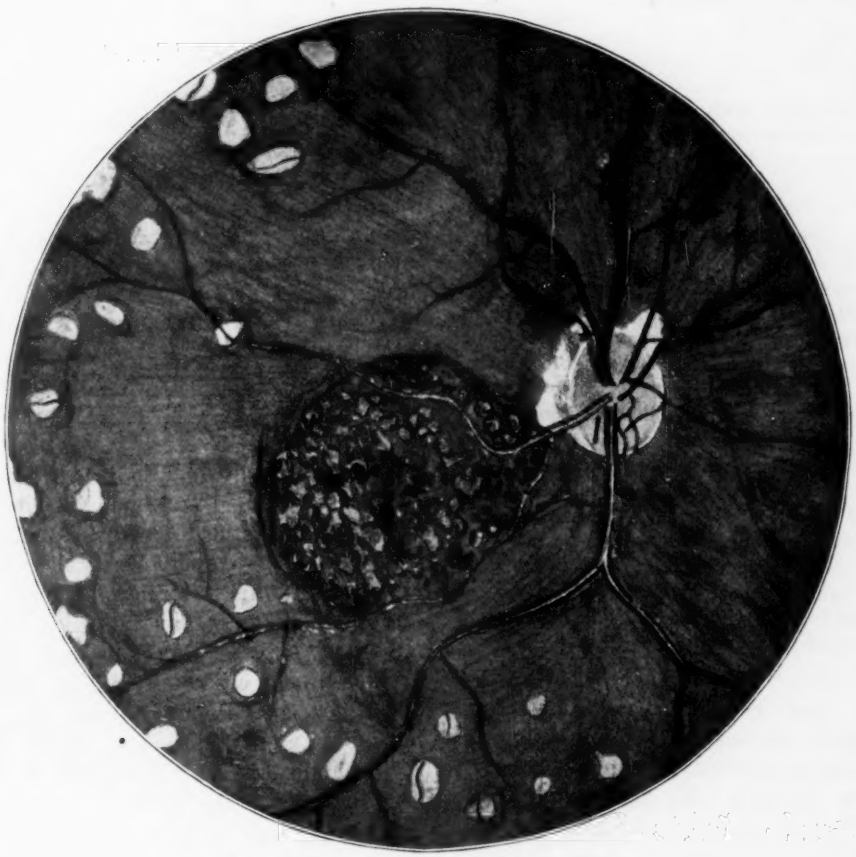

Fig. 3.—The fundus of the patient's eye.

Of more than casual interest was the appearance of the eyegrounds, which were almost identical (fig. 3). The background of the fundus in each eye was a peculiar light orange color, giving off a glowing effect. Certain areas were relucent, with the light passing through the eye proper as is seen in albinism. This

effect could be obtained from any part of the room, the light striking the fovea being directed back into the examiner's eye. No dilation of the pupil was required for the production of this phenomenon. In the right eye the nerve was elongated vertically and was rectangular rather than oval. It was somewhat less pink than normal, with a lack of marginal delineation which was due not to blurring of the disk but rather to depigmentation in the region of the retinal pigment at the papilla, so that the nerve head blended into the surrounding tissue. This effect was due to three scleral lesions surrounding the nerve head, which were actually regions of scleral atrophy. They were slightly whiter than the portion of the nerve head adjacent to them but blended harmoniously into the optic nerve with slight demarcation. The vessels at the disk were not unusual. The physiologic cup was invisible. In the macular region there were several round and oval slate-colored, pigmented lesions varying in size from ⅛ to ⅓ disk diameter. These were closely packed together, with some suggestion of overlapping. These lesions occupied an area equal to 3 or 4 disk diameters. Among the lesions were some retinal areas with a granular stippling; these areas appeared fairly normal.

When the periphery of each eyeground was scanned it was observed that the background of the fundus in the periphery was much darker than the paracentral zone. Scattered throughout the periphery in an incomplete girdle were many small grayish lesions which for the most part appeared to be old. These discrete lesions varied in size from ⅛ to ½ disk diameter. Many were gray-white and without a coating of pigment. Others contained pigment within their centers. There were minute lesions with rings of pigment. The larger grayish oval lesions had the long axis running in a horizontal direction. Through the structure of many the choroidal vessels were visible as a striking feature. Many of these foci were closely crowded and clustered into clumps of three or four. These appeared for the most part in a dirty, brownish gray background. There was a tendency toward coalescence of these patches.

Laboratory and Other Tests.—The blood chemistry was as follows: nonprotein nitrogen, 21 mg. per hundred cubic centimeters; sugar, 95 mg.; urea nitrogen, 10 mg.; cholesterol, 160 mg.; uric acid, 2 mg.; globulin, normal. The sedimentation rate was 6. The Wassermann and Kahn reactions were negative.

Examination of the urine showed a specific gravity of 1.023. Analysis of the urine gave negative results for sugar and for albumin. The microscopic study showed an occasional epithelial cell, some mucous shreds and some crystals of calcium oxalate.

The spinal fluid was clear. It contained 3 leukocytes per high power field, all of which were lymphocytes. The sugar content of the spinal fluid was 62.5 mg. per hundred cubic centimeters. There was no globulin. The Wassermann and Kahn reactions were negative with all dilutions. The colloidal gold curve was 1122210000.

Repeated tests for ova in feces disclosed the presence of Trichuris trichiura. Several tests for occult blood gave positive results at the same time.

The Mantoux test gave a strongly positive reaction after forty-eight hours, whereas the reaction to the Volmer patch test was negative.

Roentgenograms of the chest, the heart and the mediastinum were reported as showing no pathologic conditions. There was no evidence of hilar tuberculosis or of cardiac disturbance. The basal metabolic rate was + 4 per cent.

On the initial examination, tension as measured by the Schiøtz tonometer was 17 in the right eye and 21 in the left.

Treatment and Course.—The usual instillation of atropine sulfate solution along with application of hot boric acid compresses was given three times daily to both eyes. Foreign protein in the form of boiled milk was given every forty-eight hours for six injections in gradually increasing doses, starting with 4 cc. and working up to 10 cc. When the ova of T. trichiura were discovered in the stool an antihelminthic in the form of hexylresorcinol was prescribed, and several courses of therapy were outlined until successive examinations of stools failed to reveal the parasite.

The treatment for the Vogt-Koyanagi syndrome has been diversified, but since the cause is not known, most treatment has been given empirically and symptomatically. Salicylates, arsenicals, iodides, foreign proteins, tuberculin and uveal pigment have all been tried with no apparent success. Nakamura [4] administered tuberculin in a case of Vogt-Koyanagi syndrome and believed he had a beneficial form of therapy, but his patient's final visual results were limited to the ability to see movements of the hand at 6 cm. with each eye.

Verhoeff and Irvine [5] in 1936 reported an effective treatment for sympathetic ophthalmitis. A series of 35 cases was reported in which diphtheria antitoxin was used, with good results when the patients were treated early. Some time later horse serum was substituted for the original diphtheria antitoxin. Having seen fairly good results from this type of therapy, I decided to try a similar form of treatment in this case of the Vogt-Koyanagi syndrome.

4. Nakamura, B., cited by Rados, A.: Bilateral Uveitis with Associated Detachment of Retina, Arch. Ophth. **26**:543 (Oct.) 1941; Bilateral Uveitis Associated with Detachment of the Retina (Harada's Disease), ibid. **23**:534 (March) 1940.

5. Verhoeff, F. H., and Irvine, S. R.: New York State J. Med. **36**:63, 1936.

The patient was referred to the department of allergy, where careful investigation was made into his history, with special emphasis on sensitivity to horse serum. The patient was tested with concentrated horse serum, and a 4 plus reaction was reported; with a 1:10 dilution a 1 plus reaction was obtained. It was decided to desensitize the patient to horse serum by giving gradually increasing doses, beginning with very small amounts, over a long period. This was done over a period of weeks with careful check on the systemic reaction, the amount of the dose being gradually built up to a strength from which a shock reaction might be expected. The doses and the recorded reactions were listed by the allergy department in chronologic order as follows:

5/ 5/43	0.1 cc. of 1:10 dilution.....	No reaction
5/ 6/43	0.3 cc. of 1:10 dilution.....	No reaction
5/ 7/43	0.5 cc. of 1:10 dilution.....	No reaction
5/ 8/43	0.8 cc. of 1:10 dilution.....	No reaction
5/ 9/43	0.1 cc. of concentrated serum.....	No reaction
5/10/43	0.2 cc. of concentrated serum.....	No reaction
5/11/43	0.35 cc. of concentrated serum.....	No reaction
5/12/43	0.6 cc. of concentrated serum.....	No reaction
5/13/43	1.0 cc. of concentrated serum.....	No reaction
5/14/43	1.75 cc. of concentrated serum.....	Severe local reaction
5/18/43	1.75 cc. of concentrated serum.....	No reaction
5/21/43	2.0 cc. of concentrated serum.....	No reaction
5/25/43	3.0 cc. of concentrated serum.....	A little soreness
5/27/43	4.0 cc. of concentrated serum.....	A little soreness
5/31/43	5.0 cc. of concentrated serum.....	No reaction
6/ 3/43	5.0 cc. of concentrated serum.....	No reaction
6/ 5/43	5.0 cc. of concentrated serum.....	No reaction
6/ 7/43	5.0 cc. of concentrated serum.....	No reaction
6/ 9/43	5.0 cc. of concentrated serum.....	No reaction
6/11/43	5.0 cc. of concentrated serum.....	No reaction
6/14/43	5.0 cc. of concentrated serum.....	No reaction
6/16/43	6.0 cc. of concentrated serum.....	No reaction
6/18/43	7 cc. of concentrated serum.....	Patient became sick 2 hours after injection; he had dizziness, then nausea and lost his appetite for about 2 days
6/21/43	5 cc. of concentrated serum.....	Some nausea rest of day
6/24/43	5 cc. of concentrated serum.....	Some nausea rest of day
6/28/43	5 cc. of concentrated serum.....	Some nausea rest of day
6/30/43	5 cc. of concentrated serum.....	Some nausea rest of day
7/ 2/43	5 cc. of concentrated serum.....	Some nausea rest of day

The patient's pupils had been dilated with atropine sulfate in 1 per cent solution and kept under the influence of the cycloplegic for several months. His vision constantly decreased, reaching 20/400 in the right eye and 20/800 in the left eye in about four weeks. At the same time the corneal precipitates showed a marked increase in number, size and area of distribution. The "fluffs" and exudates on the capsule of the lenses appeared more pronounced. The tension, determined on March 9, 1943, was 17 for the right eye and 21 for the left (Schiøtz). About eight weeks after hospitalization it was noted that the right eye which had originally had fewer keratic precipitates on the cornea, had now become much more involved, so that the fundus was barely perceptible, whereas the left eye had begun to show some slight but definite improvement as regards both the number of keratic precipitates and the visual acuity. This observation was recorded when the amount of horse serum injected into the patient had reached 2 cc.

On May 10, 1943, in the patient's tenth week of hospitalization, it was noted that many new flocculi were visible on the inner circle of the iris of the right eye. Tension at that time was 18 (Schiøtz) in each eye; visual acuity was 20/800 in each eye.

On May 15, 1943 a severe reaction occurred locally from the injection of 2 cc. of horse serum. The patient's arm became swollen, red and painful several hours after he received the serum. The other reactions abated in twenty-four hours, but the swelling did not disappear completely until five days later. At the time of the reaction there appeared to be even more "K.P.'s" on the cornea of the right eye. The inner circle of the iris took on a new appearance, being translucent, as in senile atrophy of the iris, rather than the normal dark color. The numerous flocculi were persistent. No change was noted in the left eye at this date. After a few days the patient was again given injections of horse serum in slowly graduated doses, and by June 1 he was receiving 5 cc. without showing any serious reaction.

On June 16, 1943 it was noted that the keratic precipitates were still extremely numerous on the cornea of the right eye but that the left eye was continuing to clear up. Vision on this date was 20/400 in the right eye and 20/800 in the left, unimproved by glasses.

The patient had been desensitized to 6 cc. of horse serum, and there had been no recurrence of the original alarming reaction. The eyes were reported as being about the same. On June 18, 1943 the patient was given 7.5 cc. of horse serum. There followed within two hours a marked systemic reaction, which persisted for two days. There were dizziness, nausea and anorexia.

On July 1, 1943 it was noted that the right eye was beginning to respond to therapy; the corneal precipitates were becoming sparser, the fundus was beginning to be more visible and some of its distinctive characteristics could now be made out. The patient's vision was now much better: 20/70 in the right eye and 20/100 in the left, unimproved by glasses. There were no flocculi on the iris.

The use of atropine was discontinued in August. By this time the patient's vision had

become much better. He was able to read newspaper type with each eye. The corneal precipitates were slowly melting away; few were now visible in the left eye under the slit lamp. Many were still present on the cornea of the right eye, but these had the appearance of ice rapidly dissolving.

By the early part of September the vision in the right eye had been corrected to 20/20 and that in the left eye to 20/25. Only an occasional precipitate could be seen on each cornea. The inner circle of the iris in each eye was atrophic. The eyegrounds were the same as originally described, no changes having taken place in that region during all these months of observation.

During this period of hospitalization the patient lost 3 pounds (1.35 Kg.). The hexylresorcinol medication was continued until May.

TABLE 1.—*Results of Hematologic Examinations*

Date	Red Blood Corpuscles	White Blood Corpuscles	Hemoglobin	Monocytes	Basophils	Eosinophils	Myelocytes	Juveniles	Stab	Seg.	Lymphocytes
February 26.....	3,970,000	8,000	85%								
March 3.....	5,050,000	7,100	85%	6	..	10	..	..	3	48	33
March 5.....	4,050,000	6,050	90%	14	..	6	..	..	18	49	13
March 11.....	4,150,000	8,300	85%	6	1	11	..	..	7	49	25
March 17.....	4,700,000	8,850	95%	8	..	4	..	..	2	56	30
April 6.....	.....	6,600	90%	3	..	2	..	..	1	68	26
April 27.....	3,920,000	6,450	85%	5	..	4	..	..	3	52	36
May 17.....	4,300,000	8,500	85%	13	..	5	..	..	4	54	24
May 24.....	4,300,000	6,900	90%	10	..	6	..	..	4	58	2
May 31.....	4,750,000	8,200	85%	11	..	2	..	..	5	60	22
June 14.....	4,490,000	6,100	90%	8	..	5	..	..	6	59	22
June 28.....	4,180,000	5,550	85%	7	..	7	..	..	6	58	22
July 16.....	4,710,000	8,100	95%	8	..	9	..	..	..	52	31
September 18.....	4,800,000	7,400	90%	7	..	3	..	..	..	66	24

Several examinations of stools that failed to reveal parasites were reported in May. The studies of the blood, however, showed persistent eosinophilia until September (table 1).

DISTINCTIVE OCULAR CHARACTERISTICS IN THIS CASE

There are several ocular characteristics in this case which appear to be rather distinctive and which deserve special consideration as regards the ocular phase of the syndrome.

1. The red reflex was seen from almost any angle when a beam of light struck the eye. The effect was much like that seen in an albino (external albinism). This phenomenon was a direct result of a second feature, namely,

2. The depigmentation of the fundus, which in turn led to the albinotic appearance of the eyeground (internal albinism). There are only 4 histologic reports of cases of this disease, but these studies [6] have shown that there is a decrease in the number of chromataphores of the choroid, which tend to be grouped in the outer layers of the choroid. The pigment epithelium of the retina also shows areas of absent and of increased grouping of pigment. This change gives the fundus a particularly light appearance. This feature is striking, since the syndrome occurs in darkly pigmented races.

3. Another notable feature of this case was the characteristic arrangement of the keratic precipitates. At the height of the uveitis these precipitates were for the most part in the midcorneal zone, closely crowded together. There was no suggestion of a generalized distribution or of a specific triangular "base-down" arrangement on the cornea.

4. The occurrence of small, fluffy "Koeppe nodules" was another feature of the inflammatory phase of the syndrome, particularly when considered in association with the capsular exudation.

5. The atrophy of the inner circle of the iris in each eye is another point of special consideration, since the depigmentation of this area seems to be the most outstanding feature in the entire syndrome. In the 4 cases reported histologically little emphasis was placed on this atrophic inner circle of the iris. Matsuoka, after performing histologic studies, stated that the iris showed some thickening and some infiltration of round cells with partial destruction of the pigment epithelium. Ogawa [7] noted some scar tissue in the iris, although infiltration of round cells was also observed.

When the iris was transilluminated in the present case, a perfect ring of light was perceived

6. Matsuoka: Acta Soc. ophth. japon. **37**:1339, 1933.
7. Ogawa: Acta Soc. ophth. japon. **38**:1005, 1934.

through the inner circle of the area of the iris. This ring was not "scalloped," as in senile atrophy of the iris, nor was it "chewed out," as in heterochromic cyclitis. The small accordion-like nodular convolutions of the inner circle of the iris were completely depigmented. There was no elevation of this portion of the iris above the next, more peripheral, portion.

6. In the periphery of the retina in each eye were several atrophic spots which had certain features that seemed to make them unique. Most of these white atrophic patches were oval or egg shaped, the long axis running horizontally. The absence of bands of pigment around the patches was particularly interesting, even though in some lesions a ring of pigment might be present and though pigment might even be found within the exudate. Many of these oval pigmented exudates were crowded closely together within a field of dirty brown retina. The presence of deposits of pigment within the centers of the lesions and the absence of girdles of pigment appear to be one of the retinal features of this and allied conditions.

7. Reports by several authors of extremely deep anterior chambers were confirmed in this case. Apparently, however, the depth of the chamber was not due to any retraction of the diaphragm of the lens of the iris, nor was there any suggestion of posterior funneling of the pupil.

8. The nerve head in this case was also most unusual in appearance. It was elongated ovally, and because of several small areas of depigmentation just adjacent to it, it produced an almost oblong effect. These depigmented foci blended gradually into the nerve head, with only a slight border of separation. Not long ago, while this patient was under observation, another patient for whom a diagnosis of sympathetic ophthalmia had been made was examined. This patient had been under the care of Dr. A. C. Woods and stated that he had been treated with uveal pigment for several months. Vision had been improved from perception of light to 20/70 in each eye. On examining this patient's fundi, I saw so striking a resemblance between his eyegrounds and those of the patient whose case is presented here that successive examinations of both patients' fundi were made. In both cases the nerve heads were oblong, the fundi were depigmented and the peripheries showed identical oval white patches without pigment. These white patches have been regarded as at least suggesting sympathetic choroiditis,[8] while trauma as opposed to nontrauma has been suggested as differentiating sympathetic disease from both the Vogt-Koyanagi syndrome and Harada's disease.

9. The picture of the macula also calls for emphasis, since its heaped-up arrangement, its appearance of recurrent deposition and its density would lead one to believe that little central vision could be obtained. Vision of 20/20 and 20/25 therefore seems extraordinary and proves the necessity of attempting refraction before formulating an opinion as to the cause of reduced vision. The appearance of this heaped-up pigment, which looked as if there had been one layer deposited after another, is in line with the grouping of pigment seen in histologic sections in this and related conditions.

10. The appearance of the vitreous under the slit lamp, which unquestionably took its characteristics from the presence of uveitis in a depigmented choroid, has already been discussed as a phenomenon of special significance.

EXTRAOCULAR CHARACTERISTICS

As regards the extraocular manifestation of this syndrome, the associated signs have been thoroughly discussed by several authors[9] and require no repetition except for some statistical and some individual observations. A review and a tabulation of the case reports will disclose the incidence of these associated signs (table 2).

ETIOLOGY OF THE VOGT-KOYANAGI SYNDROME

In reports of this syndrome etiology has occupied a large place in the various discussions. Syphilis and tuberculosis have both had their advocates as causes of the disease, but an analysis of the statistics fails to offer adequate support for incriminating either. An endocrine disturbance has been postulated because of the involvement of dermal, pilous and pigmentary structures. The role played by the endocrine system in the disturbance of pigment in Addison's disease, in the condition of the skin and the hair

8. Erdmann, P.: Klin. Monatsbl. f. Augenh. **49**: 129, 1911. Givner, I.: Harada's Disease, Arch. Ophth. **30**:331 (Sept.) 1943.

9. (*a*) Parker, W. R.: Am. J. Ophth. **14**:577, 1931; Severe Uveitis with Associated Alopecia, Poliosis, Vitiligo and Deafness, Arch. Ophth. **24**:439 (Sept.) 1940. (*b*) Rones, B.: Uveitis and Dysacousia, Alopecia and Poliosis, Arch. Ophth. **7**:847 (June) 1932. (*c*) Vogt: Klin. Monatsbl. f. Augenh. **44**:242, 1905. (*d*) Koyanagi, Y.: Klin. Monatsbl. f. Augenh. **82**: 194, 1929. (*e*) Davies, W. S.: Uveitis with Associated Alopecia, Poliosis, Vitiligo and Deafness, Arch. Ophth. **14**:239 (Aug.) 1935. (*f*) Hutchinson, J.: Arch Surg., London **4**:357, 1892-1893. (*g*) Zenker, C.: Klin. Monatsbl. f. Augenh. **102**:429, 1939. (*h*) Lund, S.: Acta ophth. **16**:414, 1938. (*i*) Wexler, D.: Arch. Ophth. **57**:393, 1928.

TABLE 2.—*Analysis of Cases of the Vogt-Koyanagi Syndrome*

Case No.	Author	Year Reported	Sex	Age	Alopecia	Dysacousia	Vitiligo	Poliosis	Onset	Complication	Wassermann Test	Tuberculosis	Vision
1	Vogt	1906	Male	18	+ ?	—	—	+ 12 wk.	?	Uveitis severe	?	?	4/200; 3/200
2	Gilbert, V.	1910	Male	38	Alopecia	No dysacousia	1 vitiligo	+ 1 poliosis	—	K. P.; hyphema	—	—	6/12; 6/6
3	Erdmann	1911	Male	44	?	?	+ (—2 yr.)	+ (—2 yr.)	—	Night blindness; albinoid fundus; typical history	—	—	5/36; 5/12
4	J. Komoto	1911	Female	33	— (?)	—	+	+ (+ 3 wk.)	Headache; dizziness; vomiting; fever	Choroiditis	?	?	6/12; 6/12
5	Koyanagi	1914	Male	34	+ 3 wk.	++ 3 wk.	—	++ 3 wk.	?	Typical attack	—	?	6/36; finger counting 10 ft.
6	Koyanagi	1914	Male	34	++ 3 wk.	++ 3 wk.	—	++ 3 wk.	?	Typical attack	—	?	Finger counting 10 ft.; 6/24
7	Demaria	1917	Male	18	?	?	+ 3 yr.	+ 1	Pain in eye	Cataracts	—	—	Light perception each eye
8	Demaria	1917	Female	39	..	—	+ (1 yr.—)	+ (—1 yr.)	—	Cataract	?	?	Light perception; no light perception
9	Tsuji	1918	Male	26	+ 6 wk.	—	+	+ (+ 12)	Negative	Deep anterior chamber; secondary glaucoma	—	?	Finger counting 10 ft.; finger counting 15 ft.
10	Komoto	1918	Female	42	+ (+ 8)*	—	+	+ (+ 8)	Negative	Typical	4+	?	Finger counting 6 ft.; finger counting 6 ft.
11	Komoto	1918	Male	32	+ (+ 12)	—	+	+ (+ 12)	Negative	Typical	2+	?	Hand movements each eye
12	Arisawa	1919	Male	26	+ (+ 8)	Tinnitus (41)	—	+ (+ 8)	Cold and fever	Retinal detachment	—	?	Hand movements each eye
13	Hata and Harada	1919	Male	24	+ (+ 8)	++ (+ 1)	+	++ (+ 8)	Headache; pains in joints	Typical	—	?	0.2; 0.3
14	Komoto	1920	Female	31	+ (+ 6)	++ (+ 1)	+	—	Headache	Typical	?	?	0.3; 0.3
15	Komoto	1920	Female	31	+ (+ 12)	—	—	+ (+ 12)	Negative	Typical	—	?	0.5; 0.4
16	Baldino	1920	Male	25	..	—	+ (+ 1 yr.)	+ (+ 1 yr.)	Pain in eye	Typical	+	?	Hand movements each eye
17	Maseda	1920	Male	47	+ (+ 6)	—	+	+ (+ 6)	Severe headaches	Typical	—	?	Finger counting 6 ft.; finger counting 10 ft.; later 20/15 in each eye
18	Wexler	1928	Male	45	..	—	+ —15 yr.	+ (?)	Negative	Typical	—	?	20/200; hand movements
19	Koyanagi	1929	Male	40	+ (+ 3)	++ (+ 1)	—	+ (+ 12)	Headache; cold	Typical	—	?	Light perception each eye
20	Koyanagi	1929	Female	34	+ (+ 12)	++ (+ 1)	+	+ (+ 14)	Headache; sleepiness	Retinal detachment	—	?	20/40; 20/40+
21	Koyanagi	1929	Male	34	+ (+ 6)	++ (+ 1)	+	+ (+ 12)	Headache	Typical	—	?	0.4; 0.5
22	Koyanagi	1929	Female	40	+ (+ 1)	++ (+ 1)	+	+ (+ 16)	Headache; dizziness	Typical	—	?	Finger counting 12 ft.; finger counting 12 rt.
23	Kuhlmann	1929	Female	30	..	Tinnitus ?	—	+ (+ 12)	Headache; dizziness	Typical	—	?	Light perception

* The numbers in parentheses represent the number of weeks after onset.

24	Gilbert	1930	Female	52	—	—	+ (?) + (?)	—	?	Typical	—	?	?; ?
25	Gilbert	1930	Female	40 ?	—	—	+ (?)	—	?	Typical	—	?	?
26	Fialho	1930	Male	13	+ (+ 16)	—	?	+ (+ 16)	?	Typical	—	—	?
27	Parker	1931	Male	18	+ (+ 11)	—	+ (+ 6)	+ (+ 10)	Pain in eyes; photophobia; dimness V. A.	Typical	—	—	Finger counting 3 ft. each eye
28	Parker	1931	Female	25	+ (+ 12)	Tinnitus (+ 2)	—	+ (+ 12)	Vertigo	Typical	—	—	Hand movements
29	Westkamp	1932	Male	34	+ (+ 8)	++ (+ 1)	—	+ (+ 8)	Headache; shock	Retinal detachment	?	?	Light perception
30	Rones	1932	Male	10	—	—	+ (+ 4)	+ (+ 8)	—	Typical	— (+ uveal pigment)	—	Blind both eyes
31	Rones	1932	Female	28	+ (+ 16)	+ (+ 16)	—	+ (+ 16)	Pain in eyes	Cataract	— (uveal pigment)	—	Blind
32	Avalos	1932	Female	42	..	—	—	+ (+ 8)	Headache; vertigo; vomiting	Cataract; glaucoma	1+	?	Blind
33	Nakamura	1932	Female	28	+ (+ 10)	++ (+ 2)	—	—	Headache; pain in eyes	Retinal detachment	?	++	Finger counting 6 in.
34	Avalos	1932	Female	34	+ (+ 16)	+ (+ 2)	+ (+ 16)	+ (+ 16)	Earache; headache; pain in eyes	Cataract; glaucoma	1+	?	Blind
35	Jess	1932	Female	13	—	—	+ ?	+ (+ 16)	?	Adiposogenital dystrophy; cataract	?	—	Light perception
36	Davies	1935	Male	31	+ (+ 6)	+ (+ 4)	—	+ (+ 6)	Headache and lethargy	Fundus; no details	?	—	Light perception
37	Krasnoff	1935	Male	42	—	?	+ ?	+ ?	?	Typical	+	+	? Very poor
38	Anastasi	1936	Male	16	—	—	+ ?	+ ?	Acute articular rheumatism	Typical	—	—	Finger counting; 1/10
39	Luo	1936	Male	31	+ (+ 3)	Tinnitus (+ 4)	+ ?	+ (+ 3)	Headache; vertex, earache	Glaucoma	—	—	Light perception
40	Avalos	1936	Female	27	+ ?	+ ?	—	+ ?	Pain in eyes	Typical	?	?	Blind
41	Yanes Ferrer	1938	Female	46	+ (+ 8)	+ (+ 1)	—	+ (+ 8)	Vertigo; malaise	Phthisis bulbi	—	+1	Light perception
42	Yanes Ferrer	1938	Female	35	+ (+ 8)	+ (+ 1)	+ (+ 8)	+ (+ 8)	Vomiting	Typical	—	—	20/30; 20/30
43	Babel	1939	Female	52	—	—	+ (— 12 yr.)	+ (+ 4)	Pain in eyes	Typical	—	—	5/30; 5/15
44	Zentmayer	1941	Female	30	—	+ (+ 1)	+ ?	+ ?	Headache; eye pressure	Retinal detachment	—	—	Hand movements
45	Carrasquillo	1941	Male	36	+ (+ 12)	+ (+ 6)	+ (+ 14)	+ (+ 12)	Malaise; headache; nausea and vomiting	Cataract	3+ (U. P. neg.)	+++	Finger counting 3 ft.; finger counting 3 ft.
46	Martinez	1941	Female	20	+ (+ 6)	+ (+ 2)	—	+ (+ 6)	Acute iritis	—	—	—	Light perception each eye
47	Campos	1942	Female	34	+ (12)	+ (14)	—	+ (13)	Usual acute onset	Glaucoma (16% eosinophilia)	—	—	Light perception each eye

in eunuchs and in the occasional relationship of exudative choroiditis in women to ovarian dysfunction has been pointed out.

The possibility of a vitamin deficiency disease [10] has received little thought and less investigation. In some studies isolated features of the syndrome have occurred, such as the grayness of hair and the loss of cilia. Some form of vitamin therapy has been used in recently reported cases, with no significant response being observed.[11]

The possibility of a virus infection as an important cause was early suggested by Gilbert,[10a] who thought that the herpes virus played some part in his first case. The South American authors,[12] primarily because at the onset of the disease symptoms suggestive of cerebral involvement are present, considered that a virus infection produces a benign lymphocytic meningitis and an associated uveal involvement. Rubino [11b] expressed the opinion that the condition is uveomeningitis due to a virus with special uveomeningotropism and also with specific melanotropism, which accounts for the pigmentary phenomenon.

In 1 case reported by Avalos [12d] a cerebral decompression was performed because of suspected increased intracranial pressure. Yanes and Ferrer [12c] expressed the view that in 1 of their cases there was roentgenologic evidence of increased intracranial pressure. These authors also favored the theory that the syndrome is caused by a virus. They cited experiments in which the virus of herpes febrilis was injected into the anterior chamber of a rabbit, producing depigmentation of the iris.

Because of clinical and histologic resemblance to sympathetic ophthalmia and because of the allergic theory offered to explain sympathetic ophthalmia, a similar etiologic relationship has been postulated for the Vogt-Koyanagi syndrome. Cramer [13] attempted to explain some of the systemic signs accompanying the Vogt-Koyanagi syndrome on the basis of Elschnig's original allergic theory offered for sympathetic uveitis. This theory is, of course, open to question, especially in view of the lack of experimental proof.[14]

Peters [15] and Koyanagi have discussed the allergic role on which this syndrome may depend. Weskamp [16] believed that liberation through destruction of uveal pigment could produce an antigen which might call forth an anaphylactic process. Antibodies possessing lytic power over pigmentary structures could then be formed.

The experiments of Riehm [17] from 1928 to 1930 are of extreme and special interest in a consideration of the cause of this syndrome. This author showed that foreign protein injected into the eyes of a rabbit produced sensitizing reactions in the fellow eye only when the rabbit was pigmented and not when it was an albino. The importance of this finding is realized only when it is recalled that the Vogt-Koyanagi syndrome always occurs in darkly pigmented persons, the disease being limited to the darkly pigmented races. Some recent work by experimenters with albino and with pigmented rabbits seems to indicate that Riehm's observations were inclusive, but these same authors appear to give somewhat inconclusive proof for their deductions.[18]

Although the extensive experimental work of Woods [19] and his associates has not been completely confirmed, there is much to indicate that allergy plays an important part in causing sympathetic uveitis. Sensitivity to uveal pigment, as manifested in cutaneous tests, has been tried in only 4 instances of the Vogt-Koyanagi syndrome; in only 1 was there a positive reaction to the tests. In the remaining 3 tests the negative reactions were explained by the fact that the disease had continued long enough in the patient for desensitization to occur. A study of the skin at the site of the injection of uveal pigment has shown a definite cutaneous response, slight in vascularity but marked in cellular

10. (*a*) Gilbert, W.: Klin. Monatsbl. f. Augenh. **48**:24, 1910; **85**:65, 1930; cited by Carrasquillo.[3] (*b*) Krasnoff: Zentralbl. f. d. ges. Ophth. **37**:121, 1937. (*c*) Jess: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:469, 1932.

11. (*a*) Martinez.[2] (*b*) Rubino, A.: Ophthalmologica **101**:321, 1941. (*c*) Campos, E.: Hospital, Rio de Janeiro **22**:113, 1942.

12. (*a*) Rubino.[11b] (*b*) Campos.[11c] (*c*) Yañes, T. R., and Ferrer, O.: Rev. cubana de oto-neuro-oftal. **7**:5, 1938. (*d*) Avalos, E.: ibid. **1**:242, 1932; cited by Martínez.[2] (*e*) Koyanagi.[9d]

13. Cramer, E.: Klin. Monatsbl. f. Augenh. **51**:205, 1913.

14. (*a*) Rados, A.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **19**:579, 1913. (*b*) von Szily, A.: Klin. Monatsbl. f. Augenh. **56**:79 and 197, 1916.

15. Peters, A. A.: Klin. Monatsbl. f. Augenh. **50**: 433, 1912.

16. Weskamp, R. L.: Arch. de oftal. de Buenos Aires **7**:728, 1932.

17. Riehm, W.: Klin. Monatsbl. f. Augenh. **81**:867, 1928; **90**:477, 1933.

18. Schlaegel, T. F., Jr.: Am. J. Ophth. **27**:137, 1944.

19. Woods, A. C.: New York State J. Med. **36**:67, 1936; in Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology, London, Butterworth & Co., Ltd., 1940.

activity, which is unquestionably identical in histologic appearance with the picture of sympathetic uveitis.[20] It is conceded that the development of pigmentary allergy is not the sole cause and that some other factor is essential, such as the introduction of a virus which through the meninges eventually reaches the choroid. This may be the spark necessary to ignite an allergic phenomenon in an unusual soil. In the treatment of postoperative and post-traumatic uveitis (proved histologically to be nonsympathetic), Woods has been able to desensitize with uveal pigment patients who were sensitive to the uveal pigment tests and to produce complete arrest of the uveitis. Analysis of all reported cases shows that 20 patients had some form of cephalalgia at the onset, and it is possible that in other patients this condition was not observed or evaluated. If some acute onset could be established in all cases and if studies of the spinal fluid could be made at the incipiency of the disease, the spinal fluid might yield the essential factor necessary to complete the cycle, notwithstanding Koyanagi's statement that the spinal fluid is never under pressure and yields no etiologic factor on investigation. Should the pia-arachnoid be the seat of original involvement and should there be extension to the choroid from this embryologically related membrane, the nature of the onset of the disease might be readily understood. Once dispersion of the uveal pigment had occurred, the subsequent anaphylaxis could well be established. The experiments of Takahashi [21] and Tagami [22] bear out these suppositions, for these authors by injection of cerebrospinal fluid, vitreous, subretinal fluid and other substances from patients with Harada's disease reproduced this clinical entity in rabbits, with histologic verification, or at least simulation, of sympathetic ophthalmia.

RELATION TO HARADA'S DISEASE

That this disease and Harada's disease are related entities cannot be doubted. There have been attempts to place the two diseases in one group,[23] and other attempts to classify the syndromes into single entities.[14a]

Both Rados and Koyanagi stated that with uveitis, poliosis and alopecia there is no formation of cataracts and that the presence of cataract is a point in favor of the condition's really being Harada's disease. In Carrasquillo's analysis [3] of 29 cases 7 cases were reviewed in which complicated cataract was listed.[24] In Koyanagi's series (16 cases) there were no complicated cataracts. Rados also states that when retinal detachment occurs with nontraumatic bilateral uveitis the syndrome is specifically Harada's disease, in which "alopecia and poliosis do not figure as an integral part." Again, an analysis of all reported cases reveals that there were 5 instances of the Vogt-Koyanagi syndrome in which retinal detachment occurred.[25] Poliosis and alopecia were present in 4 of these cases.

Analysis of the entire series of cases of the Vogt-Koyanagi syndrome certainly indicates that many features which are supposedly pathognomonic for Harada's disease are present in the Vogt-Koyanagi syndrome. These features actually bridge the gap between the two diseases and probably represent different degrees of involvement in persons of variable sensitivity, the final pictures being similar. Among these features are complicated cataract, retinal detachment and depigmentation. It would therefore seem plausible that in one condition an immunity is soon developed to a severe allergic reaction (Harada's disease), whereas in the second condition the allergic process is much less acute, with the immune response almost nonexistent except when stimulated.

A summary of the tabulated case reports reveals the following facts (table 2):

Sex incidence was almost equal: There were 23 males and 24 females.

The youngest patient was 10 years old; the oldest patient was 52 years old; most patients were between 20 and 40 years of age.

Alopecia occurred in 31 of 40 recorded cases—a percentage of 73. There was no record concerning it in 7 cases.

Poliosis occurred in 43 of 47 cases—a percentage of 90. Records of 4 cases failed to include this sign.

Vitiligo occurred in 30 of 47 cases—a percentage of 63.

20. Friedenwald, J. S.: Am. J. Ophth. **17**:1008, 1934.

21. Takahashi, M.: Acta Soc. ophth. japon. **35**:114, 1931. Okamura: ibid. **41**:679, 1937; **42**:196, 1938; cited by Rados.[1]

22. Tagami: Acta Soc. ophth. japon. **35**:114, 1931.

23. Rubino.[11b] Martínez.[2] Magitot, A., and Dubois-Poulsen, A.: Ann. d'ocul. **177**:145, 1940. Avalos.[12d] Koti and Kotsuki, cited by Rados.[14a] Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3.

24. Rones.[9b] Avalos.[12d] Carrasquillo.[3] Jess.[10e] Demaria: Arch. de oftal. hispano-am. **17**:355, 1917.

25. Weskamp.[16] Koyanagi.[9d] Nakamura.[4] Zentmayer, W.: Severe Uveitis with Associated Alopecia, Poliosis, Vitiligo and Deafness, Arch. Ophth. **27**:342 (Feb.) 1942. Arisawa: Nippon Ganka Zasshi **23**:700, 1919; cited by Koyanagi.[9d]

Dysacousia was recorded in 24 of 44 cases—a percentage of 54.

Cephalgic symptoms were present in 21 cases, pain in the eyes in 11, dizziness in 6, vomiting in 4, lethargy in 2, articular pain in 2 and earache in 1.

As regards sequelae and complications, cataract occurred in 7 cases, glaucoma in 5, retinal detachment in 5 and a typical malignant terminus in 32. Endocrine dysfunction was present in 5.

Reactions to the Wassermann test were positive in 7 cases, negative in 31 and unknown in 9. The Mantoux test elicited positive reactions in 4 cases, negative in 17 and undetermined in 26. Thirty patients had vision of 3/200 or less in each eye. Only 7 patients had vision better than 20/50 in only one eye. Uveal pigment was used only a limited number of times.

SUMMARY

This report emphasizes the ocular rather than the extraocular features of this syndrome, although the extraocular features are tabulated, reviewed and listed. The incidence of each of the pathognomonic signs of the syndrome is given, all cases presented in the available literature being reviewed.[26]

Horse serum is suggested as a form of therapy.

The relationship between this syndrome and other diseases is pointed out, and an explanation of the nature of the syndrome is suggested.

26. T. H. Luo in the *Chinese Medical Journal* (**50**: 1409, 1936) referred to 10 cases in Japanese literature which were mentioned to him by Koyanagi, but Luo's reference to the Japanese literature is not correct.

MERIDIONAL ANISEIKONIA AT OBLIQUE AXES

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Many patients experience difficulties in wearing astigmatic corrections. Primarily, asthenopic symptoms are reported, but annoying distortions of objects in space are also mentioned. As a rule, the spatial distortions disappear in a short time when the correction is worn constantly. The asthenopic symptoms, on the other hand, may persist and even be so severe as to force the patient to abandon the glasses altogether. This is particularly true when the axes of the required cylinders are oblique.

The ocular discomfort has been attributed to the distortion of the retinal images produced by the cylindric spectacle lenses. These distortions have their principal effect in binocular vision. At an early date Wadsworth,[1] Culbertson [2] (who tried to correct the spatial distortion by rotating the cylinders) and Lippincott [3] described some of the changes in the position and form of objects caused by astigmatic corrections. However, they did not explain the phenomena correctly. Green [4] showed that the same effects could be obtained by using unequal figures in the stereoscope and concluded correctly that the phenomena were due to the stereoscopic interpretation of a changed relationship between the retinal images resulting from the cylindric spectacle correction. Friedenwald,[5] Koller [6] and, again, although later, Lippincott [7] expressed agreement with this. Lippincott even suggested the use of these phenomena as a test for stereoscopic vision.[8] In this test the subject would be asked to describe the apparent form of an object when viewed through a weak cylindric lens placed before one eye.

The distortion produced by a cylindric lens—or, for that matter, a meridional magnifying size lens—is an elongation of the image in the meridian of power, or magnification. Associated with that elongation are small rotary displacements of the images of all lines in space not parallel with or perpendicular to the axis of the lens. Figure 1 illustrates this fact. The image of a circular object (fig. 1 *A*), a wagon wheel for example, becomes an elliptic figure. Owing to the rotary displacements, the images of the spokes will no longer be spaced equally but will be crowded toward the meridian of elongation (magnification).

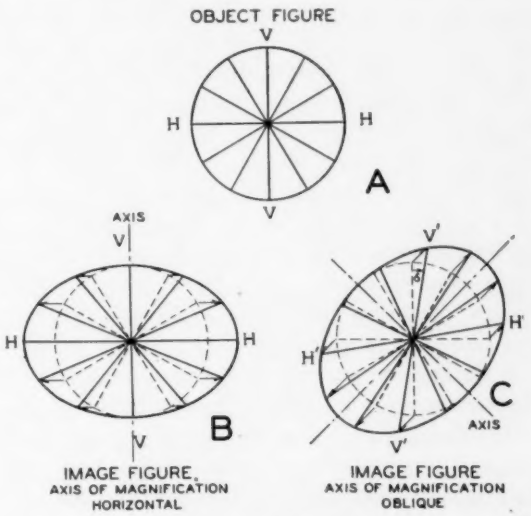

Fig. 1.—An illustration of the distortion of a wheel-like figure (*A*) when observed through a cylindric lens, (*B*) when the axis is vertical and (*C*) when the axis is oblique. A rotary deviation of the images of vertical lines occurs when the axis is oblique.

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Read before the Section on Ophthalmology at the Ninety-Fourth Annual Session of the American Medical Association, Chicago, June 14, 1944.

1. Wadsworth, O. F.: On the Effect of a Cylindrical Lens, with Vertical Axis Placed Before One Eye, Tr. Am. Ophth. Soc. **2**:342, 1879.

2. Culbertson, H.: Binocular Astigmatism, J. A. M. A. **11**:622 (Nov. 3) 1888; Am. J. Ophth. **5**:117, 1888.

3. Lippincott, J. A.: On the Binocular Metamorphopsia Produced by Correcting Glasses, Arch. Ophth. **18**:18, 1889.

4. Green, J.: On Certain Stereoscopical Illusions Evoked by Prismatic and Cylindrical Glasses, Tr. Am. Ophth. Soc. **5**:449, 1888-1890.

5. Friedenwald, H.: Binocular Metamorphopsia Produced by Correcting Glasses, Arch. Ophth. **21**:204, 1892.

6. Koller, C.: Ueber eine eigenthümliche Sorte dioptrischer Bilder: Ein Beitrag zur Theorie der Cylinderlinsen, Arch. f. Ophth. **32** (pt. 3):169, 1886; The Form of Retinal Images in the Astigmatic Eye, Tr. Am. Ophth. Soc. **6**:425, 1892.

7. Lippincott, J. A.: On the Binocular Metamorphopsia Produced by Optical Means, Arch. Ophth. **46**: 397, 1917.

8. Lippincott, J. A.: New Tests for Binocular Vision, Tr. Am. Ophth. Soc. **5**:560, 1888-1890.

If a cylindric spectacle lens is placed before one eye, with axis vertical (fig. 1 *B*), the elongation of the image will be in the horizontal meridian. The change in the disparities of the retinal images of the two eyes for all objects in space will be wholly in the horizontal meridian, and the spatial distortion will be symmetric above and below the visual plane. The small rotary deviations of the images will not cause additional stereoscopic spatial distortion. If the axis of the cylindric lens is oblique (fig. 1 *C*), the meridian of magnification is also oblique; and there will be changes in the disparities of the images in the two eyes both horizontally and vertically, these disparities increasing above and below the visual plane. They will introduce small rotary deviations in the images of vertical objects, and the result will be a distinct stereoscopic spatial distortion. This stereoscopic effect is the same as the one obtained in the stereoscope when two vertical lines, one seen by each eye, are fused. If these two lines are rotated slightly to converge at the top, the fused image appears to be a line inclined in space, nearer the observer at the top; if the two lines diverge at the top, the fused image appears to be tipped away from the observer at the top. In other words, whenever there is a deviation from parallelism between the images of a vertical line in the two eyes, that line will appear to be inclined with the top toward or away from the observer. We have designated the rotary deviation between the retinal images of the two eyes producing this steroscopic effect as the declination error,[9] following and extending the terminology introduced by Stevens.[10]

Declination errors can be produced by optical means, as has just been shown, by placing cylindric lenses at oblique axes in front of the eyes. They can also be produced by a torsion of the two eyeballs, relative to each other, about the visual axes. When introduced optically, the declination error varies for the different meridians of the retinal image, the maximum occurring when the meridian of power is 45 or 135 degrees (fig. 1 *C*). If due to a cyclophoria, the declination error is the same for all meridians of the retinal images. Torsions of the eyeballs, therefore, cannot correct the declination errors produced optically. They could correct the error only in one meridian. Finally, declination errors may result if there is a basic meridional aniseikonia at oblique axes not due to the correction of astigmatic refractive errors.

Declination, as a possible cause of asthenopic symptoms,[11] at one time were much discussed in the American ophthalmologic literature. Controversy developed about this point and about the relation of the declinations to cyclophoria and the part played by the oblique muscles of the eyes in correcting them.

But the discussion had no practical consequences, and interest in the subject soon waned. In more recent times the problem has been given little, if any, clinical attention. However, when it is mentioned, it is generally implied that spatial distortions can be the seat of ocular discomfort. Wolffberg,[12] for example, cited a number of cases in which patients reported annoying distortions of their surroundings and were unable to wear corrections for their astigmatism in spite of prolonged attempts. For most of these patients the axis of astigmatism was oblique. Erggelet[13] recognized the problem and described and interpreted it correctly. He, too, pointed out that some patients with high cylindric corrections at oblique axes would rather put up with the lowered visual acuity than wear the cylindric lens. But he could offer no suggestion as to how the distortions could be avoided. He stated, however, that the spectacle wearer may learn how to reinterpret the visual impressions and how to disregard the distortions. This is sometimes made easier if the cylindric lenses are first placed in the horizontal or the vertical meridian and are then, over a period of weeks, slowly turned to the proper axis.

No solution has yet been found for the problem of the distortions arising in binocular vision from the wearing of cylindric spectacle lenses at oblique axes and the discomfort experienced by the patient. The reason is that the problem was not approached from the proper viewpoint. The declination errors at once become accessible to measurement and correction when they are recognized as being an aspect of aniseikonia.

Whenever ametropias of refractive origin of different amounts in the two eyes are corrected

9. Ogle, K. N., and Madigan, L. F.: Astigmatism at Oblique Axes and Binocular Stereoscopic Spatial Localization, Arch. Ophth. **33**:116 (Feb.) 1945.

10. Stevens, G. T.: A Treatise on the Motor Apparatus of the Eye, Philadelphia, F. A. Davis Company, 1906.

11. Eaton, F. B.: The Physiology of Certain Oculo-Motor Phenomena with Respect to Some Recent Theories of Asthenopia, J. A. M. A. **23**:329 (Sept. 1) 1894. Howe, L.: The Muscles of the Eye, New York, Putnam's Sons, 1907, vol. 2, pp. 264 and 364. Savage, G. C.: Ophthalmic Myology, ed. 2, Nashville, Tenn, 1911.

12. Wolffberg: Störung des perspektivischen Sehens durch binokular korrigierende Zylindergläser, Central-Ztg. f. Optik **35**:10 and 20, 1914.

13. Erggelet, H.: Die Refraktion und die Akkommodation mit ihren Störungen, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 2, p. 673.

by glasses, an aniseikonic error may be expected.[14] Thus, the correction of unequal or unsymmetric astigmatism would theoretically result in a meridional aniseikonic error.

The correctness of this assumption has been borne out by innumerable measurements with the standard eikonometer in cases of astigmatic anisometropias in the horizontal and vertical meridians. Until recently there was no instrumentation which would permit an easy determination of the amount of aniseikonia in oblique meridians. This problem has been solved by the construction of the Ames space eikonometer. This instrument utilizes the characteristic spatial distortions produced by the aniseikonic errors in binocular vision. Using the space eikonometer, Ogle and Madigan [9] were able to demonstrate that there is, indeed, a relationship between the declination error produced by the oblique astigmatism and the incorrect spatial localization of a binocularly seen vertical line. In the majority of cases a relationship was found between the declination errors computed [15] on the basis of the existing astigmatism and the declination errors measured by the amount of the spatial distortion. The incorrect appearance of objects in space can be corrected by offsetting the measured declination error with meridional magnifying lenses at oblique axes.[9]

It was of importance to investigate to what extent patients would benefit from the correction of the declination errors by equalizing the ocular images. The present paper is a report on the results obtained in a first group of patients for whom were prescribed aniseikonic corrections at oblique axes based on measurements with the space eikonometer.

Before we describe the method of measurement employed and the results obtained, one more point must be discussed.

It still seems to be the general opinion that the incorrect binocular spatial orientation due to the deformation of the retinal image by spectacle lenses, in particular cylindric lenses, while capable of causing ocular discomfort, usually disappears with continued wearing of the lenses, especially in young people.[16] This opinion, based on ophthalmologic experience, implies that the aniseikonic error which gave rise to the spatial distortion also disappears.

However, aniseikonic errors produced by corrections for anisometropia can often be measured in patients who have worn their glasses for many years and who ordinarily notice no spatial distortions. Nevertheless, the spatial distortions corresponding to aniseikonic errors can be demonstrated under appropriate experimental conditions.[17] The presence of a declination error of the calculated amount in patients who have worn their glasses for as long as fifty years has been found by Ogle and Madigan.[9]

Furthermore, the study of one of us (H. M. B.)[18] on the effect of prolonged wearing of meridional size lenses on spatial orientation certainly suggests that only a small fraction, if any, of the image size differences introduced by the lenses disappears. Whether or not the incorrect spatial localization caused by the aniseikonic error will be manifest depends on the absence or presence of strong perspective clues in the person's surroundings. In the presence of strong perspective clues, such as are contained in the normal surroundings of all city dwellers, the stereoscopic stimuli which would lead to an incorrect binocular spatial localization soon become ineffective.

The fact that a patient is unaware of any spatial distortion is not evidence that aniseikonic errors do not exist. We shall revert to this point once more when discussing the results obtained with patients wearing oblique aniseikonic corrections.

INSTRUMENTS AND PROCEDURE

Instruments.—A meridional aniseikonic error at an oblique axis can be determined if, in addition to the usual measurements of the image size differences in the horizontal and vertical meridians, the vertical declination error can be found. As was pointed out, the Ames space eikonometer [19] makes these measurements possible. In previous papers it has been shown that a good correlation exists between the aniseikonia

14. This is not to imply, however, that all aniseikonic errors arise in the correction of refractive anomalies or that the magnitude of the aniseikonia expected on the basis of the refractive error will be measured (Burian, H. M., and Ogle, K. N.: A Study of the Aniseikonia in a Case of Increasing Unilateral Index Myopia, Am. J. Ophth. **26**:480, 1943).

15. The declination error can be computed on the basis of the astigmatism present if the physical dimensions of the glasses, the eye wire distance and the keratometer readings are known. For details, see Ogle and Madigan.[9]

16. Hofmann, F. B.: Raumsinn des Auges, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 2, Berlin, Julius Springer, 1925, vol 3, chap. 13, pp. 109-112. More recently, Jackson, E.: Practical Importance of Aniseikonia, Am. J. Ophth. **26**:18, 1943.

17. Ogle, K. N.: Association Between Aniseikonia and Anomalous Binocular Space Perception, Arch. Ophth. **30**:54 (July) 1943.

18. Burian, H. M.: Influence of Prolonged Wearing of Meridional Size Lenses on Spatial Localization, Arch. Ophth. **30**:645 (Nov.) 1943.

19. Ames, A., Jr.: The Space-Eikonometer Test for Aniseikonia, Am. J. Ophth. **28**:248, 1945.

measured in the horizontal and vertical meridians on the standard eikonometer and that measured on the space eikonometer [17] and, also, that the declination error can be measured on the space eikonometer.[9] The space eikonometer, therefore, was used to determine the oblique aniseikonic prescriptions given the patients considered in this study.

The Space Eikonometer.—The essential parts of the space eikonometer (fig. 2) are (1) a system of selected test elements; (2) suitable test magnifying lens units, by which the size of the images in the two eyes can be changed optically, and (3) a head rest.

The test elements consist of (*a*) an oblique cross made up of two smooth-stretched cords at right angles to each other and (*b*) two pairs of smooth vertical plumb cords, erected two in front and two behind the cross.

The oblique cross, stretched from the corners of a 5 foot (152 cm.) square frame, is set up vertically at a distance of 3 meters from the subject's eyes. The plane of the cross is carefully erected at right angles to the subject's objective median and visual (horizontal) planes. In the plane of the cross, and through its center, a plumb line is suspended.

Two of the vertical plumb cords are suspended 60 cm. in front of, and two 60 cm. behind, the cross. The cords in front of the cross are separated about 50 cm., and they thus subtend a visual angle of approximately 12 degress to the observer. The cords are equidistant from the subject's objective median plane, and the plane of each set of cords is parallel with his frontal plane. Suitable apertures restrict the subject's binocular field of view entirely to the test elements and also prevent him from seeing the ends of the cords. In order to aid in their identification, the cords of the cross are red; the vertical cords in front are green, and the central cord through the cross and the two rear vertical cords are white. The test elements are illuminated by four Lumiline lamps placed so as to produce no shadows. The elements are seen against a uniformly black background.

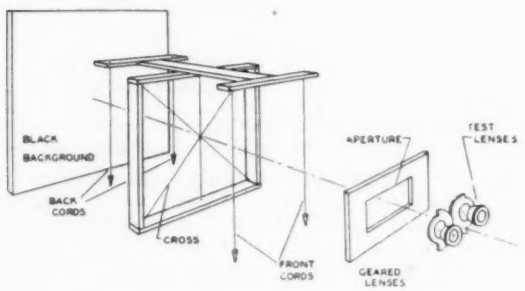

Fig. 2.—Perspective drawing of the essential parts of the space eikonometer.

The test magnification lens system (fig. 3) consists of two parts: 1. Two pairs of meridional adjustable size units which measure up to 4.00 per cent,[20] one pair before each eye. One pair is fixed at axis 90 (to change the size of the retinal image in the horizontal meridian) and the other at axis 180 (to change the size of the retinal image in the vertical meridian). The magnifications of these lenses then can be adjusted to equalize the horizontal and vertical components of the aniseikonic error in either eye. 2. A matched pair of 2.00 per cent meridional size lenses, one before each eye, are geared together so that they rotate in equal amounts but in opposite directions. These lenses permit one to offset the vertical declination error caused by a meridional aniseikonic error at an oblique axis by introducing a declination error in the opposite direction without disturbing the horizontal or vertical image size relationships. The two lenses can be rotated, and

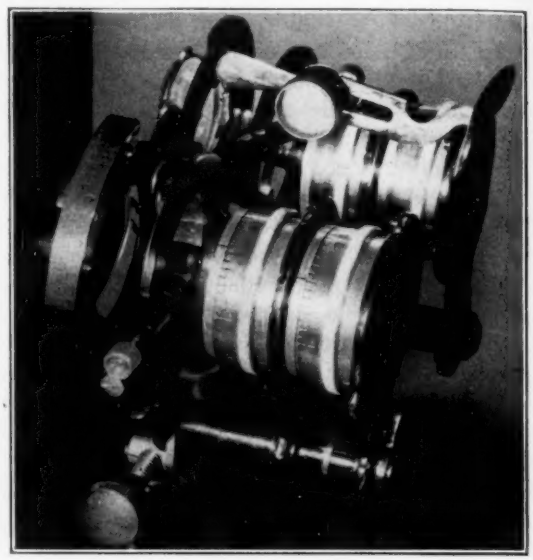

Fig. 3.—Photograph of the test lenses on the space eikonometer, showing the adjustable size units for axis 90 and axis 180 measurements, and the geared meridional size lenses by which the declination errors between the two eyes can be measured.

the degree of rotation is indicated on a scale. When the axes of the two lenses are parallel (and vertical), the magnification of one lens offsets the magnification of the other, and their combined binocular effect is zero. If the two lenses are rotated through a small angle, their axes converge upward or downward and introduce, or correct, a vertical declination of the images of the two eyes. The vertical and the horizontal component of the two lenses offset one another. The scale is calibrated to read directly the declination angle (δ) in degrees.[21]

The head rest supports the head and keeps the eyes of the subject properly centered before the instrument.

Procedure.—If a difference exists in the size of the ocular images in the two eyes, or is introduced by size lenses, in the horizontal meridian only, one of each pair of the vertical cords will appear nearer the subject than does the other. Also, the oblique cross will appear rotated about a vertical axis. The nearer cord and the nearer side of the cross will be on the side of the eye having the smaller ocular image. By increasing the size of the image in this eye in the horizontal meridian by means of the appropriate magnifying test

20. Ogle, K. N.: An Optical Unit for Obtaining Variable Magnification in Ophthalmic Use, J. Optic. Soc. America **32**:143, 1942.

21. The relationship between the vertical declination angle, δ, and the angle of rotation of the geared lenses, ρ, is given by the formula $\delta = 0.29\ m \sin 2\ \rho$, in which m is the per cent of magnification of the meridional size lenses (2.00 per cent in the present instrument).

lens, the cords and the two sides of the cross can be made to appear equidistant from the observer.

If a difference in the size of the ocular images exists, or is introduced by size lenses, only in the vertical meridian, the cross alone will appear rotated about a vertical axis (the vertical cords will be unaffected), the farther side being on the side of the eye having the smaller image. By magnifying the image in that eye in the vertical meridian by means of the appropriate test lens, the two sides of the cross can be made to appear the same distance from the subject, i. e., frontoparallel. Thus, the presence and magnitude of a horizontal and a vertical image size difference can be detected and measured by the difference in the apparent rotation of the vertical plumb lines and the cross. When both a horizontal and a vertical image size difference exist, the horizontal one must be corrected first.

If a meridional image size difference is present at an oblique axis, so that there is a resulting declination error in the ocular images of vertical lines, then the plane of the cross appears inclined (as though rotated about a horizontal axis through the center of the cross).[22] If the upper part appears inclined away from the observer, the axes of the correcting meridional size lenses must be rotated so as to converge upward toward the median plane (forehead) of the subject in order to correct that inclination. The measured declination error, δ_m, is said to be positive in this case. If the upper part of the cross appears inclined toward the subject, the axes of the correcting meridional size lenses must be rotated so as to converge downward toward the median plane (chin) of the subject in order to correct that inclination. The measured declination error, δ_m, is said to be negative in this case. By rotating the geared lenses by the proper amount and in the proper direction, the apparent inclination of the plane of the cross can be varied until it and the central vertical cord, through the center of the cross, appear in the same plane. The degree of rotation of the geared lenses measures the vertical declination error.[23] The incorrect apparent orientation of any one of the test elements during the test may affect the relative orientation of the others, so that the final result must be determined in successive approximations by correcting in sequence the horizontal and the vertical image size differences and then by correcting the declination errors.

The data are obtained in the same manner as are the data with the standard eikonometer, namely, by questioning the subject regarding the apparent positions of the test elements as the examiner makes different adjustments of the test lenses. Here, too, the method of limits within which the test elements appear correctly oriented is applied to obtain the final correction. An estimate of the stereoscopic sensitivities of the patient to the three parts of the test is given by one-half the range within which he reports the vertical cords and the cross to be properly oriented.

The final aniseikonic correction follows directly from the measurements in a straightforward manner. It can be obtained by referring to prepared tables.[24] The validity of the correction is usually checked on the space eikonometer, trial magnifying lenses being used.

RESULTS AND COMMENT

Aniseikonic corrections at oblique axes were prescribed for a total of 76 patients. Nearly all these patients had an astigmatic correction of at least 0.50 D. cyl. in one eye, the axis deviating from the horizontal and the vertical by at least 15 degrees. The ages of the patients ranged from 17 to 70 years, the average age being about 39 years. The patients have worn the aniseikonic corrections at least nine months; the majority have worn them over two years.

On receiving their correction, the patients were asked to report their reactions to the lenses after having worn them for one month and then, again, later. After the study had been in progress for one and a half years, a questionnaire was sent out. A large proportion of the patients have been reexamined since they received their glasses.

In attempting to evaluate the results obtained thus far, one must keep in mind that the patient material in this study was highly selected. The asthenopic symptoms of the patients were, on the whole, much more severe than those of the average patient. Many of the patients had tried, with more or less success, numerous refractive, prismatic and even standard aniseikonic corrections. The oblique aniseikonic correction was tried with several patients only as a last resort. With some of the patients other ocular

22. Under ordinary circumstances one would expect the cross alone to appear vertical, while the vertical cords should appear inclined, instead of the reverse. Experiment has shown, however, that the apparent angle between the vertical central cord and the apparent position of the plane of the cross is the same in either case (Ogle, K. N.: Theory of the Space Eikonometer, to be published).

23. The direction of the calculated correction for the declination error, δ_c, in a given case will be positive if the axes of the correcting minus cylinders converge upward, and it will be negative if they converge downward.

24. The tables are based on the following theory: Any given aniseikonic correction consists of an over-all magnification, o%, combined with a meridional magnification f%, at axis ϕ. The formula, then, for the component of that correction in the horizontal meridian (axis 90) is $h = o + f \sin^2 \phi$, and the formula for the component in the vertical meridian (axis 180) is $v = o + f \cos^2 \phi$. The correction for the declination error between the two eyes will be given by the formula $\delta = 0.29\ f \sin 2\phi$.

If, as a result of measurement h, v and δ are known, o, f and ϕ can be found. One has for ϕ the formula $\tan \phi = 3.5\,\delta/(v\text{-}h)$. The meridional per cent of magnification, f%, will be given by the formula $f = [(v\text{-}h)^2 + (3.5\,\delta)^2]\ ½$, or $f = 3.5\,\delta/\sin 2\phi = (v\text{-}h)/\cos 2\phi$. Then the per cent over-all magnification, o%, will be given by the formula $o = ½\ (v + h\text{-}f)$.

Under certain conditions the correction can be transformed so that the axes of the meridional corrections will coincide with the axes of astigmatism of the two eyes. In general, this consists of an over-all correction together with a meridional correction for each eye. (Ogle, K. N.: Meridional Magnifying Lens Systems in the Measurement and Correction of Aniseikonia, J. Optic. Soc. America **34**:302, 1944).

Tabulation of Data and Results Obtained on Patients to Whom Oblique Aniseikonic Prescriptions Were Given

Case No.	Age, Yr.	Chief Complaints	Correction Worn *	Correction Given *	Visual Acuity	Stereopsis, % †	Phoria ‡ Distant Vision	Phoria ‡ Near Vision	Percentage Aniseikonia § Axis 90	Percentage Aniseikonia § Axis 180	Measured Declination δ^m Degrees	Error Calculated δ^m Degrees	Equivalent Aniseikonic Prescription, %	Results
1	40	Frontal headaches	−0.50 −0.25 x180 −0.25 −0.25 x5	−0.62 −0.37 x30 −0.37 −0.25 x60	20/15 20/15	100	Ortho	4½△ X	R 1.25 ± 0.25	R 0.50 ± 0.50	+ 0.20 ± 0.10	+ 0.11	R 1.25 x60	Improvement in headaches
2‖	42	Severe eyestrain, frontal headaches	+1.25 −1.50 x30 +1.00 −1.25 x180	+1.75 −1.50 x40 +1.50 −1.25 x170	20/15 20/15	100	1△ X ½△ RH	4△ X ½△ RH	L 0.25 ± 0.25	L 0.75 ± 0.25	− 0.54 ± 0.20	+ 0.54	R 0.50 x125 L 2.00 x40	Marked relief
3	33	Ocular discomfort, photophobia, nervousness	+0.50 −0.50 x25 −0.25 −0.37 x170	+0.75 −0.75 x25 +0.25 −0.50 x165	20/20+ 20/20+	100	¼△ X	6△ X	R 1.00 ± 0.25	L 0.25 ± 0.25	+ 0.20 ± 0.10	+ 0.23	L 1.00 x120	No benefit
4	30	Ocular fatigue, headaches	−0.87 −0.25 x45 −0.87 −0.25 x135	−1.00 −0.25 x40 −1.00 −0.75 x115	20/20+ 20/20+	No record	Ortho	Slight X	L 1.00 ± 0.50	L 0.25 ± 0.25	+ 0.29 ± 0.10	+ 0.39	R 0.50 x60 L 1.25 x120	Marked relief
5	48	Eyestrain	+1.25 −3.50 x10 +0.25 −2.62 x164	+1.00 −3.25 x10 +0.50 −2.75 x170	20/20 20/20	100	6½△ X	2△ X	L 0.00 ± 0.37	R 2.75 ± 0.50	+ 0.20 ± 0.16	+ 1.00	R 2.50 x10 L 1.00 x90	Marked benefit from correction
6	31	Severe eyestrain and disabling headaches	−0.75 −1.25 x155 +0.75 −1.00 x177 R 2.50%x180	−0.75 −1.25 x155 +0.75 −1.00 x177	20/40 20/30	90	2△ S	2-4△ S	R 0.50 ± 1.50	R 2.00 ± 1.00	− 0.20 ± 0.20	− 0.39	R 2.50 x150	Definite relief from oblique axis correction
7	67	Eyeache and headaches, nervous tension	+1.00 −5.00 x100 +1.25 −4.50 x75 R 1.50%x90 L 1.25%x180	+1.50 −5.50 x100 +2.00 −5.00 x73	20/20 20/20−	No record	¾△ S	5△ X	R 1.50 ± 0.75	L 1.75 ± 0.62	− 0.85 ± 0.40	− 1.45	R 1.25 x100 L 1.50 x63	Increased relief with oblique axis correction
8	62	Severe headaches and ocular discomfort	−1.00 −4.00 x145 3△B.U. +3.00 −1.50 x125 3△B.D. R 1.50%O.A. ⌒ 4% x 145 **	−0.50 −4.00 x143 3△B.U. + 1△B.I. +3.50 −1.50 x120 3½△B.D.	20/40# 20/15	100	2½△ S	5△ X 1△ LH	R 2.50 ± 1.50	R 4.50 ± 1.50	− 1.12¶ ± 0.38	− 0.82	R 1.50 O.A. ⌒ 4.50 x143	Marked comfort from oblique axis correction
9‖	18	Marked ocular fatigue, frontal headaches in close work	+0.12 −0.12 x180 −0.25 sph.	Plano Plano	20/15 20/15	100	Ortho	1△ X	R 0.75 ± 0.50	R 0.50 ± 0.50	− 0.38 ± 0.20	0.00	R 1.50 x135	Marked relief
10	25	Constant headaches, ocular fatigue, inability to read even for few minutes	+0.75 sph. +0.75 −0.25 x7	+1.50 −0.25 x45 +1.50 −0.25 x135	20/15 20/15	100	½△ X	4△ X	R 0.50 ± 0.25	R 0.50 ± 0.25	+ 0.30 ± 0.10	+ 0.28	R 1.50 x45	Excellent result; patient able to work 10-12 hours a day
11	52	Blurring, headaches, associated with use of eyes, photophobia	0.00 −0.37 x110 0.00 −0.25 x5	0.00 −0.37 x20 +0.37 −0.37 x45	20/15 20/15	100	1△ S	2½△ X	R 0.50 ± 0.25	R 0.50 ± 0.25	− 0.20 ± 0.12	− 0.21	R 0.75 x120	Partial relief
12	18	Blurring of vision, headaches after reading or movies	+0.75 −0.75 x175 +5.75 −4.75 x15 Correction worn only occasionally	+1.25 sph. +6.00 −5.00 x15	20/15 20/20+	100; slow	Slight S	2△ X	R 2.75 ± 1.00	0.00 ± 1.00	− 0.72 ± 0.28	− 1.05	R 3.00 x105 L 1.00 x115	Definite benefit
13‖	41	Severe eyestrain and irritation, photophobia, headaches	+1.00 −3.25 x168 1△B.U. +1.25 −2.25 x40 2△B.D.	+1.00 −2.75 x163 1△B.U. +1.00 −2.25 x40 2△B.D.	20/15 20/15	100	1△ S 3△ LH	5△ X 2½△ LH	R 0.50 ± 0.50	R 2.50 ± 0.50	− 1.30 ± 0.20	− 1.70	R 2.25 x143 L 0.50 x53	No relief
14	57	Ocular fatigue, photophobia, frontal headaches	+1.00 −5.00 x72 +1.00 sph. R 0.75%x90 L 1.50%x180	+1.00 −5.00 x72 +1.50 −0.50 x80	20/40 20/25	100	½△ X	4△ X	R 0.75 ± 0.25	L 1.00 ± 0.25	+ 0.55 ± 0.20	+ 0.95	R 2.00 x72 L 1.50 x170	Partial relief
15	23	Headaches, eyestrain	+1.25 −1.25 x15 +1.25 −1.00 x175 R 1.00% O.A.	+1.25 −1.25 x15 +1.25 −1.00 x175	20/15− 20/15−	100	Slight X	1△ X	R 1.00 ± 0.25	R 0.25 ± 0.37	+ 0.29 + 0.10	+ 0.29	R 1.50 x60	Definite benefit from oblique axis correction
16	34	Extreme eyestrain, inability to use eyes, nausea, nervousness, distortion of space and form	+0.37 −3.50 x22 +0.25 −3.50 x170	+1.00 −4.00 x15 1½△B.O. +1.00 −4.50 x162.5 1½△B.O.	20/20+ 20/20+	100	2△ S††	6-7△ S	L 0.25 ± 0.25	L 0.50 ± 0.50	+ 0.96 ± 0.24	+ 1.62	R 1.75 x45 L 2.00 x135	Marked relief

17	43	General ocular distress, inability to wear glasses, headaches	+2.25 —3.75 x177 +1.00 —3.75 x149	+2.00 —3.50 x180 +1.75 —4.00 x150	20/20 20/25	100	Ortho	7△ X	R 2.25 ± 0.50	0.00 ± 0.50	+ 0.75 ± 0.20	+ 1.32	R 2.50 O.A. ⌒ 2.00 x150	Symptoms partially relieved; patient able to wear glasses constantly
18	38	Severe headaches, photophobia, difficulty in space perception, nervousness	—5.00 —1.25 x19 —5.00 —0.50 x167	—4.00 —1.25 x180 2½△B.I. —4.00 —0.50 x150 2½△B.I.	20/40+ 20/40+	100	1△ X	12△ X	R 1.85 ± 1.00	R 0.50 ± 1.00	— 0.46 ± 0.38	+ 0.29	R 3.00 x115	Marked benefit
19	29	Ocular fatigue, headaches, blurring vision, photophobia	—2.75 —0.50 x130 —1.25 —0.50 x50 R 1.50%x90	—2.50 —0.75 x145 —1.50 —0.25 x60	20/15 20/15	No record	5△ X 1△ RH	10△ X	0.00 ± 0.37	L 0.25 ± 0.50	+ 0.29 ± 0.20	— 0.42	R 0.50 x55 L 0.75 x150	Marked relief, but evidence not convincing
20	48	Eyestrain and headaches.....	+0.75 —0.75 x95 +0.75 —0.37 x45	+2.00 —0.75 x70 +1.75 —1.00 x40	20/15 20/15	100	1△ S	4△ X	R 0.35 ± 0.25	L 0.75 ± 0.50	— 0.20 ± 0.20	— 0.27	L 1.00 x23	Marked relief
21	46	Ocular irritation, nervousness, photophobia	+2.50 —2.50 x180 +2.25 —2.75 x10 R 0.75%x90 L 1.50%x180	+2.50 —2.50 x180 +2.00 —2.50 x18	20/20 20/15	100	1△ X ½△ LH	8△ X ½△ LH	R 0.25 ± 0.25	R 1.00 ± 0.37	— 0.38 ± 0.16	— 0.42	R 1.25 x45	No definite relief
22	44	Headaches and nausea when reading; eye discomfort	+1.37 —1.00 x32 +1.12 —0.75 x2	+1.50 —1.00 x40 +1.25 —0.75 x180	20/20+ 20/20+	100	½△ X	7½△ X	R 0.75 ± 0.25	L 0.25 ± 0.25	+ 0.29 ± 0.20	+ 0.35	R 1.50 x40 L 0.75 x180	Much more comfortable with new correction
23	32	Constant headaches, photophobia and eyestrain	+1.50 —0.75 x65 +1.00 —0.37 x160 R 1.00%x90 L 1.25%x180	+1.50 —0.75 x65 +1.25 —0.50 x150	20/15 20/15	100	1△ S	1½△ X	R 0.75 ± 0.25	R 0.75 ± 0.50	+ 0.20 ± 0.10	+ 0.35	R 1.75 O.A. L 1.25 x150	Partial relief
24	32	General fatigue, occipital and frontal headaches; reading difficulties	+0.50 —0.50 x20 +0.75 —0.25 x65	+0.75 —0.75 x20 +0.75 sph.	20/20 20/20	100	½△ X	4△ X	R 0.50 ± 0.25	R 2.00 ± 0.50	+ 0.29 ± 0.10	+ 0.15	R 1.50 x20	No benefit
25	49	Ocular distress, photophobia, headaches of long standing	+3.50 sph. +3.50 sph. L 1.00%O.A. ⌒ 2%x90	+4.25 —0.50 x140 +4.50 —0.50 x60	20/13 20/13	No record	2△ X ½△ LH	2△ X	L 1.50 ± 0.25	L 1.00 ± 0.25	— 0.20 ± 0.16	— 0.52	R 1.00 x145 L 1.75 O.A.	Partial relief; more comfort with oblique axis correction than any previous one
26	44	Ocular fatigue, nervous tension	+2.00 —0.25 x45 +2.00 —0.37 x120	+2.25 —0.50 x45 +2.25 —0.50 x120	20/20+ 20/15—	90; slow	4△ S	3△ X	R 0.25 ± 0.25	R 0.25 ± 0.25	+ 0.20 ± 0.10	+ 0.36	R 0.75 x45	Partial relief
27	48	Severe ocular fatigue, lacrimation, photophobia	+1.25 —1.00 x90 +1.25 —1.62 x93	+1.50 —1.25 x85 +1.50 —2.00 x95	20/15+ 20/15	100	½△ X	5½△ X	L 0.25 ± 0.25	0.00 ± 0.25	+ 0.38 ± 0.20	+ 0.24	R 0.50 x35 L 1.00 x125	Definite help from glasses
28	43	General ocular distress and irritation, reading difficulty, headaches	+0.25 —0.50 x140 +0.25 —0.50 x40 L 1.00%x90	+0.50 —0.50 x140 +0.50 —0.50 x45	20/15 20/15	100	Slight S	3△ X 1△ LH	R 0.50 ± 0.50	R 0.50 ± 0.50	— 0.38 ± 0.20	— 0.45	R 1.00 x145 L 0.50 x55	No benefit
29	55	Constant headaches	+1.25 —0.75 x122 +1.50 —0.50 x75	+2.00 —1.00 x120 +2.75 —1.00 x60	20/15+ 20/15+	100	2½△ X	6½△ X	R 0.75 ± 0.75	R 0.25 ± 0.75	— 0.38 ± 0.20	— 0.78	R 1.25 x120	Marked relief from headaches
30	50	Headaches and photophobia...	+0.87 —0.37 x60 +0.75 —0.37 x170 R 1.00%x90 L 1.00%x180	+0.87 —0.37 x40 +0.75 —0.25 x20	20/15 20/15	100	2△ S	4△ X	R 1.25 ± 0.50	R 0.25 ± 0.50	+ 0.20 ± 0.20	+ 0.11	R 1.25 x40 L 1.00 x20	Benefit from oblique axis correction
31	33	Eyestrain and eyeache, followed by sick headaches; difficulties in space perception	+1.37 —1.37 x39 +1.37 —1.37 x148	+2.25 —1.50 x30 +2.00 —1.50 x150	20/15 20/15	100	1△ X	5△ X	R 0.75 ± 0.25	R 0.50 ± 0.25	+ 0.38 ± 0.10	+ 0.96	R 1.25 x50	Definite improvement
32	48	Severe and continuous headaches, fatigue	—0.50 —0.25 x85 —0.12 sph.	—0.50 —0.50 x60 Plano —0.25 x135	20/15+ 20/15+	100	Ortho	2△ X	R 0.50 ± 0.25	L 0.25 ± 0.25	+ 0.29 ± 0.16	+ 0.08	R 1.75 x60 L 0.75 O.A. ⌒ 0.25 x30	Partial relief
33	63	Eyestrain; inability to read...	—0.62 —1.00 x155 —0.50 —2.00 x72.5	—0.62 —1.00 x155 —0.50 —2.00 x72.5	20/15 20/15	100	1△ S ¼△ RH	4△ X ¼△ LH	L 1.25 ± 0.25	L 0.50 ± 0.37	— 0.80 ± 0.20	— 0.72	R 0.75 x155 L 2.75 x72.5	Complete relief
34	29	Eyestrain	—0.25 —0.25 x60 —0.12 —0.25 x115 R 1.00%O.A.	—0.25 —0.25 x45 0.00 —0.50 x115	20/15 20/15	100	Slight S	1△ X	R 0.50 ± 0.25	R 0.75 ± 0.25	+ 0.20 ± 0.12	+ 0.30	R 1.25 x45	No benefit
35‖	56	Severe eyestrain and frontal headaches of long standing	+5.00 —1.75 x60 +4.75 —1.75 x115 L 1.25% O.A.	+5.50 —1.75 x60 +5.00 —1.75 x120	20/20—2 20/20—3	No record	3△ S	2△ X	0.00 ± 0.25	L 1.50 ± 0.25	+ 0.85 ± 0.20	+ 1.70	R 1.00 x60 L 2.50 x150	Marked relief

Tabulation of Data and Results Obtained on Patients to Whom Oblique Aniseikonic Prescriptions Were Given—Continued

Case No.	Age, Yr.	Chief Complaints	Correction Worn *	Correction Given *	Visual Acuity	Stereopsis, % †	Phoria ‡		Aniseikonia § Percentage		Declination Measured δ^m Degrees	Error Calculated δ^m Degrees	Equivalent Aniseikonic Prescription, %	Results
							Distant Vision	Near Vision	Axis 90	Axis 180				
36	48	Photophobia, ocular discomfort at near work and movies, constant headaches of long standing	+0.50 —0.50 x80 Plano	+0.75 —0.75 x80 +0.50 —0.25 x70	20/15 20/15	100	1△ X	6△ X	R 1.25 ± 0.25	R 0.50 ± 0.50	+ 0.20 ± 0.20	+ 0.15	R 0.50 O.A. ⌒ 0.75 x75	Except for photophobia, great relief
37	60	Constant frontal and temporal headaches, not associated with use of eyes	—0.25 —0.62 x97 —0.25 —0.50 x5	0.00 —0.75 x105 0.00 —0.50 x60	20/15 20/15	100	1△ S	9△ X	L 0.50 ± 0.50	L 0.50 ± 0.50	— 0.38 ± 0.20	— 0.39	L 1.00 x60	No relief from headaches
38	33	Eyestrain, especially on driving; infrequent headaches	Plano —0.75 x60 Plano —1.50 x155	Plano —0.75 x60 Plano —1.50 x155	20/15— 20/15—	100	2△ X	2△ X	R 1.25 ± 0.25	L 0.50 ± 0.50	+ 0.55 ± 0.20	+ 0.54	R 2.00 x60 L 1.00 x155	Marked relief
39	31	Severe headaches, eyestrain, reading difficulties	+0.25 —0.25 x90 +0.50 —1.75 x25	+0.25 —0.25 x90 +0.50 —1.75 x25	20/20 20/20—	100; slow	1½△ X 1½△ RH	5-6△ X 1½△ RH	R 0.75 ± 0.25	L 1.50 ± 0.50	— 0.55 ± 0.20	— 0.58	L 1.25 x25	Improvement in headaches
40	32	Need for glasses, ocular fatigue, some headaches	None	+0.62 sph. +2.50 —1.75 x55	20/15+ 20/20—	100	1△ S	Ortho	R 2.25 ± 0.25	R 2.50 ± 0.50	— 0.55 ± 0.20	— 0.88	R 1.00 O.A. ⌒ 2.00 x135	Very satisfactory
41	52	Headaches and ocular pain, ocular fatigue and nervousness, difficulties in space perception	+1.50 —1.37 x170 +1.50 —1.37 x55	+1.50 —1.50 x165 +1.75 —1.50 x55	20/15 20/15	100	1△ X	7△ X	R 0.50 ± 0.25	R 1.25 ± 0.25	— 0.38 ± 0.12	— 0.89	R 1.75 x155	No help from glasses
42	39	Headaches, reading difficulty	+2.50 —4.00 x165 —1.75 —0.75 x180	+2.50 —4.00 x165 —1.75 —0.75 x180	20/20 20/15	100	1½△ S	6△ X	L 0.50 ± 0.75	L 0.25 ± 0.25	— 0.38 ± 0.12	— 0.69	L 1.50 x45	Possibly some benefit
43	33	Headaches and eyestrain, ocular fatigue	+1.50 —1.25 x180 +0.75 —1.00 x30	+2.25 —1.25 x180 +1.25 —1.00 x30	20/15 20/20	70	Ortho	1½△ S	R 0.75 ± 0.25	0.00 ± 0.25	— 0.20 ± 0.10	— 0.26	R 0.75 O.A. L 0.75 x120	Partial relief
44‖	30	Eyeache; frontal and occipital headaches on use of eyes, of long standing; photophobia	+2.75 —3.25 x20 +3.25 —4.00 x140	+2.50 —3.50 x20 +3.25 —4.00 x140	20/15 20/15	100	1△ S ½△ LH	4△ X ½△ RH	R 1.00 ± 0.25	R 0.25 ± 0.25	+ 1.00 ± 0.20	+ 2.80	R 3.00 x50 L 1.00 x140	Marked relief
45	41	Frequent headaches, nervousness	+1.12 —0.37 x10 +1.00 —0.50 x45	+1.25 —0.37 x90 +1.37 —0.87 x60	20/20 20/20	No record	Ortho	1△ X	0.00 ± 0.50	L 0.25 ± 0.37	— 0.29 ± 0.12	— 0.37	L 0.75 x60	Definite benefit
46	30	Ocular fatigue, especially for near work; nervousness	+1.75 —0.62 x15 0.50% O.A. +2.00 —0.87 x165 1.50% x90	+1.75 —0.62 x115 +2.00 —0.87 x165	20/15—2 20/20+2	No record	1△ S	2△ X	L 0.75 ± 0.20	L 0.25 ± 0.25	+ 0.16 ± 0.18	+ 0.46	L 1.00 x15	Definite relief with oblique axis correction
47	44	General ocular discomfort and fatigue; poor reader	+1.12 —3.50 x3 +0.75 —4.00 x175 R 2.00% x90 L 0.75% x180	+1.00 —3.50 x5 +0.75 —4.00 x167	20/20 20/20	100	1△ X	6△ X	R 3.00 ± 0.50	0.00 ± 0.50	+ 0.38 ± 0.15	+ 0.80	R 3.00 O.A. L 3.00 x167	Former aniseikonic correction gave unusual relief but oblique axis correction much better
48‖	40	Frontal headaches; inability to read comfortably	—1.00 sph. —1.00 —0.25 x20 R 1.50% x90	—1.00 sph. —1.00 —0.25 x25	20/15 20/15	No record	2-3△ X	5-6△ X	R 1.25 ± 0.25	0.00 ± 0.50	+ 0.20 ± 0.10	0.00	R 1.50 x105	Considerable benefit from oblique axis correction
49‖	25	Constant ocular discomfort, frontal headaches, photophobia, space difficulties	—1.25 —3.50 x170 1△ base up —1.00 —4.00 x17 1△ base down	—1.25 —3.50 x170 1△ base up —1.00 —4.00 x17 1△ base down	20/30+ 20/30	90, with difficulty	1△ S 1△ LH	2△ X 1½△ LH	L 0.50 ± 0.25	0.00 ± 0.25	— 1.07 ± 0.40	— 1.49	R 1.50 x135 L 1.50 x45	Marked benefit

50	26	Headaches and ocular discomfort, photophobia	Plano —0.75 x165 —0.50 —1.25 x10 R 1.00% O.A. L 0.75% x180	—0.25 —0.75 x160 —0.75 —1.25 x10	20/15 20/15	70	Ortho	3△ X	L 0.50 ± 0.25	L 0.50 ± 0.25	— 0.20 ± 0.12	— 0.33	R 1.62 x165 L 1.75 x10	Some benefit from oblique axis correction
51	73	Eyestrain, continuous headaches, reading difficulties	+1.37 —0.62 x163 +2.25 —1.25 x8 R 2.50% x90	+2.00 —0.50 x15 +3.00 —0.50 x160	20/20— 20/20—	100	1△ X ½△ LH	8△ X ¾△ LH	R 3.00 ± 0.25	R 2.50 ± 0.25	+ 0.95 ± 0.20	+ 0.34	R 1.00 O.A. ⌒ 3.25 x50	Very marked relief of symptoms
52	38	Ocular fatigue, effort to read; carsickness	+0.62 —0.37 x98 +3.50 —3.50 x15	+1.25 sph. +5.00 —5.00 x27.5	20/13 20/25	100	½△ X	3½△ X	R 4.50 ± 0.25	R 0.50 ± 0.37	— 0.90 ± 0.12	— 0.95	R 4.50 x118	Marked improvement with glasses
53	41	Headaches, photophobia, eyestrain	—0.25 sph. —0.25 —0.25 x15	0.00 —0.12 x135 +0.25 —1.00 x37	20/15+ 20/15+	100	Ortho	8△ X	0.00 ± 0.25	L 0.87 ± 0.25	— 0.55 ± 0.10	— 0.60	R 0.75 x135 L 1.50 x37	Patient completely satisfied; relief
54	46	Headaches from driving and movies, reading difficulties, cannot wear glasses	+1.25 —1.00 x150 +1.25 —1.25 x60	+1.50 —1.00 x140 +1.50 —1.25 x62	20/15 20/15	100	1½△ X	4½△ X	R 0.50 ± 0.75	R 0.50 ± 0.50	— 0.30 ± 0.10	— 0.67	R 1.00 x140 L 0.75 x62	Complete relief
55	45	Continuous ocular fatigue, space difficulties, vertigo, some headaches	+4.00 —2.50 x10 +1.75 —1.25 x157	+5.00 —3.00 x15 +3.00 —1.75 x165	20/20—1 20/20—2	100	2△ S	2△ X	L 1.75 ± 0.25	L 0.75 ± 0.25	+ 0.38 ± 0.20	+ 0.74	L 1.75 x120	Partial relief
56‖	22	Patient cannot wear glasses for even few minutes; severe eyestrain and headaches	+0.50 sph. +1.00 —1.00 x27	+1.25 —0.50 x170 +3.25 —5.00 x25	20/15—2 20/30+2	None	1½△ X ¾ RH	6△ X ¾ RH	R 2.25 ± 0.75	L 0.75 ± 0.50	— 1.00 ± 0.30	— 1.59	R 2.75 O.A. ⌒ 0.75 x170 L 4.00 x25	Definite benefit, patient able to read with correction
57	48	Eyestrain and discomfort of long standing	+0.50 —0.75 x180 +4.00 —4.00 x42	+1.00 —1.00 x180 +5.00 —5.00 x42	20/20+3 20/20—2	80	Ortho	6△ X	R 2.00 ± 0.75	R 2.00 ± 0.75	— 1.12 ± 0.29	— 1.81	R 4.00 O.A. L 4.00 x42	No benefit
58	62	Ocular fatigue	+6.00 —0.50 x10 +6.50 —2.25 x60	+6.00 —0.50 x10 +6.50 —2.25 x60	20/25— 20/20	No record	3½△ X Ex-cycle 4°	18△ X	R 1.25 ± 0.25	R 0.75 ± 0.25	+ 0.56 ± 0.38	— 0.58	R 2.50 O.A. L 2.50 x150	Good results
59	33	Spatial difficulties, photophobia	+1.62 —1.12 x170 +1.75 —1.25 x10	+2.25 —1.50 x10 +2.25 —1.50 x170	20/20 20/20	100	1△ S ¼ LH	3△ X ½ LH	R 0.25 ± 0.25	L 0.25 ± 0.25	+ 0.38 ± 0.12	+ 0.47	R 2.00 x10 L 2.00 x170	Definite benefit
60	18	Ocular fatigue, frontal headaches, no help from wearing of correction, carsickness	+0.50 —0.50 x180 +2.25 —3.25 x17 Correction worn occasionally	+0.50 —0.50 x175 1△ B.D. +2.50 —4.00 x22 1△ B.I.	20/20 20/20	50, slow	4△ X	12△ X	R 1.00 ± 1.50	R 1.00 ± 1.00	— 0.55 ± 0.38	— 1.13	R 2.00 x175 L 3.00 x22	Definite relief, new correction worn constantly
61	35	Sick headaches, ocular fatigue, space distortion	+1.50 —1.75 x150 +1.00 —1.00 x25 L 1.00% O.A.	+1.50 —2.00 x155 +0.75 —1.00 x20	20/15 20/15	100	½△ X	2△ X	L 0.50 ± 0.50	L 0.25 ± 0.25	— 0.38 ± 0.20	— 1.00	R 0.75 x140 L 1.50 x50	Marked relief
62	36	Carsickness, air sickness.....	0.00 —0.25 x165 1△ B.I. —0.25 —0.25 x165 1△ B.I.	+0.50 —0.50 x145 +0.25 —0.50 x10	20/15 20/15	100	½△ S	4△ X	R 0.50 ± 0.25	R 0.25 ± 0.50	— 0.20 ± 0.10	— 0.19	R 0.75 x127	Results indeterminate
63	55	Ocular discomfort with use of eyes	+1.87 —0.25 x165 +1.87 —0.25 x30	+2.50 —0.25 x135 +2.50 —0.25 x45	20/15 20/15	100	½△ X ¼ RH	5△ X ¼△ RH	R 0.25 ± 0.25	R 0.50 ± 0.25	— 0.20 ± 0.10	— 0.10	R 0.50 O.A. L 1.25 x45	Results uncertain

* In this column, and in other columns, O.A. indicates an overall magnification.
† Obtained on Keystone DB[6] chart.
‡ X indicates exophoria; S, esophoria; L.H., left hyperphoria; R.H., right hyperphoria; ortho, orthophoria; cyclo., cyclophoria.
‖ Case described fully in this paper.
§ Aniseikonic measurements made while patient was wearing refractive prescription in standard Tillyer spectacle lenses.
¶ Probably inaccurate because of oblique prisms in prescription.
# Corneal scar.
** Determined by the proximation method on the standard eikonometer.
†† Exophoria prior to operation.

factors, such as convergence insufficiency, complicated the problem. Other patients were definitely neurotic. Circumstances compelled us to conduct our study on such a group of patients. Favorable results are, therefore, all the more significant.

We have refrained from a detailed statistical study and have divided the patients roughly into three groups: (1) patients who had definitely been relieved of their symptoms; (2) patients who were partially relieved of their symptoms, and (3) patients for whom the prescription resulted in failure. The data on 13 of the 76 patients had to be discarded because of insufficient information about the result (4 patients) or because definite or probable extraneous factors interfered with the evaluation (5 and 4 patients respectively).

Of the remaining 63 patients, 36 have reported marked relief from their symptoms; 16, partial relief, and 11, no relief whatever. The accompanying table lists the most pertinent data for each of the 63 patients.

We shall now discuss in some detail the results obtained and report the history in representative cases.

REPORT OF CASES [25]

CASE 1.—I. E. B., a woman aged 28, a school teacher, came for examination because of severe "eyeache," photophobia and frontal and occipital headaches on use of her eyes, especially at moving pictures, in driving and in close work. These symptoms had existed since early in life but had become increasingly severe in the past eight years. She had worn glasses constantly since the age of 12 years. Frequent medical examinations had revealed nothing abnormal. She had been under expert ophthalmic care and was operated on in 1934 and 1935 for exophoria and right hyperphoria, which were discovered by an occlusion test.

Refraction and Visual Acuity.—Visual acuity of 20/15 was obtained with a correction of + 2.75 D. sph. ⌒ — 3.25 D. cyl., axis 20 for the right eye and of + 3.25 D. sph. ⌒ — 4.00 D. cyl., axis 140 for the left eye. At 20 feet (6 meters) there were 1 prism diopter of esophoria and ½ prism diopter of left hyperphoria; at 16 inches (40.6 cm.), 4 prism diopters of exophoria and ½ prism diopter of left hyperphoria. Examination for stereopsis revealed 100 per cent depth perception with the Keystone DB 6 chart.

Measurements on Space Eikonometer.—The measurements for aniseikonia were as follows:

At axis 90; (right) 1.00% ± 0.25%
At axis 180: (right) 0.25% ± 0.25%
Measured declination error, δ_m: + 1.00° ± 0.20°
Computed declination error, δ_c: + 2.80°

The following correction was prescribed:

Right eye: + 2.50 D. sph. ⌒ — 3.50 D. cyl., axis 20 ⌒ 3.00% meridional magnification, axis 50.

Left eye: + 3.25 D. sph. ⌒ — 4.00 D. cyl., axis 140 ⌒ 1.00% meridional magnification, axis 140.

These glasses were worn as fit-overs for six months; they proved completely satisfactory. Reexamination before the correction was prescribed in permanent form yielded essentially the same measurements. The patient has now worn the permanent glasses for fifteen months and reports great relief from her symptoms except for occasional headaches, which are less severe than in the past.

This case is a typical one in which the high oblique astigmatic correction required by the patient produced a declination error which was of significant amount and in the direction expected from the astigmatic error. The patient showed a high stereoscopic sensitivity, and the correction of the aniseikonia afforded her relief from severe symptoms, of long standing.

This case does not seem to offer particular difficulties in interpretation. There are, however, cases in which the measured and the computed declination errors do not agree. Nevertheless, in some instances the patients are benefited, if the measured declination error is used as the basis for the the aniseikonic prescription.

CASE 2.—Mrs. R. H. B., a housewife aged 41, engaged in sculpturing and literary work, came for examination because of severe eyestrain, particularly in the right eye, caused by the use of the eyes for distant, as well as near, vision. If close work was continued, frontal headache developed. In bright light and sunshine, as well as in close work, the right eye tended to close. At the age of 8 years the patient had been struck in the right eye by an air rifle bullet. She had worn glasses since attending high school and had always had expert ophthalmic care. The last prescription was given her two years prior to examination at the Dartmouth Eye Institute.

The right eye showed an iridodialysis between 7 and 8 o'clock, and the pupil of that eye was accordingly distorted. Otherwise, both eyes were normal in all parts.

Refraction and Visual Acuity.—Visual acuity of 20/15 was obtained with a correction of + 1.75 D. sph. ⌒ — 1.50 D. cyl., axis 40 for the right eye and of + 1.50 D. sph. ⌒ — 1.25 D. cyl., axis 170 for the left eye. This refractive correction was identical with the one the patient was wearing except for an increase of + 0.50 D. sphere for each eye. Readings at 20 feet (6 meters) showed 1 prism diopter of exophoria and ½ prism diopter of right hyperphoria, and at 16 inches (40.6 cm.), 4 prism diopters of exophoria and ½ prism diopter of right hyperphoria. Examination for stereopsis revealed 100 per cent depth perception with the graduated Keystone DB6 chart.

25. Unless it is specifically mentioned, the eyes of these patients had clear media and no pathologic condition of the fundi. Here, δ_m designates the measured declination error, and δ_c, the declination error computed on the basis of the existing astigmatism; the values are recorded in degrees. An estimate of the stereoscopic sensitivity of the patients is given by one-half the range within which the patient reported that the vertical cords and the cross of the space eikonometer were properly oriented. Sensitivities of ± 0.25 per cent for axis 90 and axis 180 and of 0.10 to 0.20 degree for the declination error are considered normal. Aniseikonic measurements and corrections are indicated by the magnification necessary to equalize the ocular images.

Measurements on Space Eikonometer.—The measurements for aniseikonia were as follows:

At axis 90: (left) 0.25% ± 0.25%
At axis 180: (left) 0.75% ± 0.25%
Measured declination error (δ_m): — 0.55° ± 0.20°
Computed declination error (δ_c): + 0.55°

The equivalent aniseikonic correction was prescribed as follows:

Right eye: + 1.75 D. sph. ⌒ — 1.50 D. cyl., axis 40 ⌒ 0.50% meridional magnification, axis 125
Left eye: + 1.50 D. sph. ⌒ — 1.25 D. cyl., axis 170 ⌒ 2.00% meridional magnification, axis 40

The patient wore these glasses with fit-over size lenses for four months, with good results, and was then given permanent glasses after reexamination, which substantiated the findings of the first examination. When she reported again, two years and four months later, she stated that she no longer had eyestrain or headaches and that her right eye closed only occasionally in bright light. She volunteered the information that objects stood out in relief much better; trees, for instance, were no longer flat on the hill. Her only complaint was that of blurring in near vision for the past year. The measurements revealed no need for a change in the correction for distance; an addition of + 0.75 D. sph. for each eye was given for reading.

The salient feature of this case is the fact that a declination error was measured in the direction opposite that which would be theoretically expected. According to the patient's astigmatic correction (axes of minus cylinders converging upward), she should have had a declination error which would be corrected by meridional size lenses with axes converging upward (positive declination error). Actually, a negative declination error was measured. Nevertheless, excellent subjective results were obtained by correcting the measured declination error.

It is difficult to explain the discrepancy between the measured and the calculated declination error in this case. Ogle and Madigan [9] have suggested that a declination error measured in the direction opposite that expected from the astigmatism may be due either to the presence of a cyclotropia or to a basic meridional aniseikonic error, not optical in nature, which is in a direction opposite that introduced by the astigmatic correction.

While at present one can only suggest a possible explanation of the measurements obtained in this case, there are other cases which indicate the direction which future research must take in order to solve this problem. These are the cases (1) in which there is a measurable and clinically significant declination error, in spite of the fact that the patient has a spherical refractive correction or is emmetropic; and (2) in which there is a large amount of oblique astigmatic error, yet no declination error can be measured.

CASE 3.—E. J. I., a Catholic priest aged 40, complained of frontal headaches and "confusion" after reading for fifteen minutes and after driving a car for about one hour, and of inability to concentrate on his reading matter. The trouble began sixteen years ago, after a "nervous breakdown." He was given glasses five years prior to the first examination at the Dartmouth Eye Institute (correction of — 0.75 D. sph. for each eye).

Refraction and Visual Acuity.—Visual acuity of 20/15 in each eye was obtained with the following correction:

Right eye: — 1.00 D. sph.
Left eye: — 1.00 D. sph. ⌒ — 0.25 D. cyl., axis 20

At 20 feet (6 meters) there was 2 to 3 prism diopters of exophoria, and at 16 inches (40.6 cm.) 5 to 6 prism diopters of exophoria.

Examination with the standard eikonometer showed that a correction of 1.50% meridional magnification, axis 90 should be combined with the refractive correction for the right eye. The patient wore this correction for one year and reported that vision was improved with these glasses, but that there were apparent tipping of the floor and distortion of objects. Reexamination showed that the refractive error was unchanged.

Measurements on Space Eikonometer.—The measurements for aniseikonia were as follows:

At axis 90: (right) 1.25% ± 0.25%
At axis 180: 0.00% ± 0.50%
Measured declination error (δ_m): + 0.20° ± 0.10°
Computed declination error (δ_c): 0

The equivalent aniseikonic correction prescribed was as follows:

Right eye: — 1.00 D. sph. ⌒ 1.50% meridional magnification, axis 105
Left eye: — 1.0 D. sph. ⌒ — 0.25 D. cyl., axis 25

After wearing this correction for four months, the patient reported decided improvement; he was able to do several hours of concentrated reading, without the "confusion" formerly experienced; there were still headaches, but they were much fewer and less severe. After one year the improvement was still present.

In this case a declination error was measured in spite of isometropic refraction (except for insignificant astigmatism in the left eye). The results of examination with the standard eikonometer approximated the measurements with the space eikonometer. But the result obtained was much better when the axis of the meridional magnification was rotated by 15 degrees, so that a correction of the measured declination error was effected.

Even more striking is a case in which a declination error was measured in the presence of emmetropia.

CASE 4.—S. B. W., a girl aged 15 years, was a high school student when she was first seen, in 1938. For the past three years she had had severe ocular fatigue and frontal headaches when doing close work, watching moving pictures or riding in a car. Glasses prescribed eighteen months prior to examination here (right eye: + 0.12 D. sph. ⌒ — 0.12 D. cyl., axis 180; left eye: + 0.25 D. sph. ⌒ — 0.25 D. cyl., axis 5) had not given relief.

Refraction and Visual Acuity.—The right eye was emmetropic, with visual acuity of 20/15; the left eye had visual acuity of 20/15 with correction of —0.25 D. sph.

At 20 feet (6 meters) there was orthophoria; at 16 inches (40.6 cm.), 1 prism diopter of exophoria.

Measurement with the standard eikonometer revealed the need for 1.00 per cent over-all magnification of the right ocular image. The following correction was given:

Right eye: plano lens ⌒ 1.00% over-all magnification
Left eye: plano lens

The patient returned three years later, stating that the glasses had been of help for about two years but that now, again, she had had frequent frontal headaches. The results of refraction were as follows:

Right eye: —0.25 D. cyl., axis 150, vision 20/15
Left eye: —0.25 D. cyl., axis 40, vision 20/15

Examination on Space Eikonometer (performed without refractive correction).—The measurements for aniseikonia were as follows:

At axis 90: (right) 0.75% ± 0.50%
At axis 180: (right) 0.50% ± 0.50%
Measured declination error (δ_m): + 0.38° ± 0.20°
Computed declination error (δ_c): 0.

The following equivalent aniseikonic correction was prescribed:

Right eye: plano lens ⌒ 1.50% meridional magnification, axis 135
Left eye: plano lens

The patient wore this correction with great comfort. Two years and three months later she came for reexamination, at her father's wish, because she had lately experienced eyestrain at the end of the day; she is now engaged in exacting war work.

Measurement on Space Eikonometer.—With the patient's correction, the measurements for aniseikonia were as follows:

At axis 90: (right) 1.00% ± 0.50%
At axis 180: 0.00 ± 0.50%
Measured declination error (δ_m): — 0.20° ± 0.20°

Without glasses the measurements for aniseikonia were as follows:

At axis 90: (right) 1.00% ± 0.50%
At axis 180: (right) 0.75% ± 0.62%
Measured declination error (δ_m): 0.38° ± 0.20°

These measurements indicated that the declination error was somewhat undercorrected, and the following equivalent aniseikonic correction was suggested: right eye, plano lens ⌒ 1.75% meridional magnification, axis 130; left eye, plano lens. The patient did not feel that her symptoms warranted a change of glasses at this time.

If there are cases of isometropia or emmetropia with measurable declination errors in which symptoms are relieved when the declination error is corrected, it is logical to assume that there must be cases of astigmatic corrections at oblique axes in which no declination error is shown. Such cases do indeed exist, and the following case is an example.

CASE 5.—G. S. J., a woman aged 24, engaged in housework, came to the Dartmouth Eye Institute in 1937. She gave a history of "sick headaches" in her family, and she herself complained of severe frontal and temporal headaches, beginning on the left side and spreading to the right. The headaches might come at any time and last for days, and they were often completely disabling. Lately they had been accompanied with nausea and vomiting. The vomiting did not relieve the headaches. They were not necessarily connected with the use of the eyes, but her eyes had always been bad. She was subject to eyestrain, and vision was not good. She had worn glasses constantly since the age of 5 years. Because of her headaches and ocular discomfort, she had to give up school when she was a freshman in high school. She had always been under expert medical and ophthalmic care. The removal of wisdom teeth, treatment of sinuses and treatment for migraine headaches were of no avail. The last refractive correction was given nine months prior to the first examination at the Dartmouth Eye Institute.

Refraction and Visual Acuity.—The results of refraction were as follows:

Right eye: + 3.00 D. sph. ⌒ — 4.00 D. cyl., axis 25, vision 20/30
Left eye: + 2.50 D. sph. ⌒ — 4.00 D. cyl., axis 155, vision 20/30

Findings at 20 feet (6 meters) varied from orthophoria to ½ prism diopter of esophoria; at 16 inches there was 1 to 2 prism diopters of exophoria. Examination for stereopsis revealed 100 per cent depth perception with the graduated Keystone DB6 chart.

Examination on the standard eikonometer indicated the need for over-all magnification by 1.50 per cent of the ocular image of the left eye. This was incorporated with the refractive correction. The glasses did not help; neither did various refractive corrections or monocular occlusion. The patient was followed for five years; during this time numerous medical examinations, including encephalographic studies, were made, but she obtained no relief.

In January 1942 she was first examined with the space eikonometer. She proved on the whole to be a good observer, though the data showed some variability. Average data obtained in examinations with the space eikonometer over a period of days were as follows:

At axis 90: (left) 0.75% ± 0.50%
At axis 180: (left) 1.25% ± 0.62%
Measured declination error (δ_m): + 0.20° ± 0.30°
Computed declination error (δ_c): + 1.45°

Various aniseikonic corrections, on the basis of measurement and of calculation, were tried, but none afforded any relief. Six months later the refractive correction was changed somewhat (right eye, + 3.00 D. sph. ⌒ — 4.50 D. cyl., axis 22; left eye, +2.00 D. sph. ⌒ — 4.50 D. cyl., axis 157) and the measurements in the space eikonometer showed improvement in sensitivity. They were as follows:

At axis 90: (right) 0.75% ± 0.25%
At axis 180: (left) 0.50% ± 0.25%
Measured declination error (δ_m): 0.00 ± 0.20°
Computed declination error (δ_c): 1.45°

The patient wore the refractive correction only, and six months later the aniseikonic data were:

Axis 90: (right) 1.25% ± 0.25%
Axis 180: (left) 1.75% ± 0.25%
Measured declination error (δ_m): 0.00 ± 0.10°

The aniseikonic error at axes 90 and 180 was prescribed for, but the correction did not afford the patient any relief, and she soon discarded the heavy fit-over glasses.

This case cannot properly be called one of oblique meridional aniseikonia. It is cited because no declination error could be measured in spite of a large oblique astigmatic correction. Nor would it be correct to classify this case as one of failure to correct the oblique meridional aniseikonia. The seat of the patient's difficulty must be sought elsewhere.

There are, however, cases in which the prescription resulted in complete failure, although at least partial improvement might have been expected in view of the large oblique astigmatic error and the close agreement between the measured and the computed data. An illustration is the following case.

CASE 6.—F. A. Q., a dentist aged 41, complained of considerable eyestrain (soreness, pulling and smarting of the eyes) after close work, reading, driving and watching motion pictures. There were also photophobia and some headache. The headaches had decreased after a radical antrotomy was performed four years before. The patient had worn glasses constantly since the age of 17 years. The eyestrain had been increasing for the past twelve years. He had always been under competent ophthalmic care; he had had refractive corrections, with and without vertical prisms, for left hyperphoria; monocular occlusion; additional correction for near work, and examination for aniseikonia before coming to the Dartmouth Eye Institute. The aniseikonic glasses with corrections at axes 90 and 180 proved of no help; they were, in fact, less comfortable than the simple refractive correction.

Refraction and Visual Acuity.—The results of refraction were as follows:

Right eye: + 1.00 D. sph. ⌒ — 2.75 D. cyl., axis 163, vision 20/15

Left eye: + 1.00 D. sph. ⌒ — 2.25 D. cyl., axis 40, vision 20/15

At 20 feet (6 meters) there were 1 prism diopter of esophoria and 3 prism diopters of left hyperphoria, and at 16 inches (40.6 cm.), 4 to 6 prism diopters of exophoria and 2½ prism diopters of left hyperphoria. Examination for stereopsis revealed 100 per cent depth perception with the graduated Keystone DB6 chart.

Measurement with the standard eikonometer indicated the need for 2.00 per cent meridional magnification, axis 180 for the right eye. The following prescription was ordered:

Right eye: + 1.00 D. sph. ⌒ — 2.75 D. cyl., axis 163 ⌒ 1 Δ base up ⌒ 2.00% meridional magnification, axis 180

Left eye: + 1.00 D. sph. ⌒ — 2.25 D. cyl., axis 40 ⌒ 2 Δ base down

An additional correction of + 1.00 D. sph. for near work was given for both eyes. This correction did not afford relief, and eight months later measurements were made on the space eikonometer. The findings were as follows:

At axis 90: (right) 0.50% ± 0.50%
At axis 180: (right) 2.50% ± 0.50%
Measured declination error (δ_m): — 1.30° ± 0.20°
Computed declination error (δ_c): — 1.70°

An equivalent aniseikonic correction was suggested, with the addition to the patient's refractive correction of 2.50 % meridional magnification, axis 1.35 for the right eye. Examination seven and, again, sixteen months later revealed that the measured declination error was somewhat smaller, but still highly significant. The final equivalent aniseikonic correction arrived at was as follows:

Right eye: + 1.00 D. sph. ⌒ — 2.75 D. cyl., axis 163 ⌒ 2.25% meridional magnification, axis 143

Left eye: + 1.00 D. sph. ⌒ — 2.25 D. cyl., axis 40 ⌒ 0.50% meridional magnification, axis 53

An additional correction of + 1.00 D. sph. for near vision was given for each eye. This correction was no more satisfactory than were the previous ones, and the patient has since reported that he gets along best with an old refractive correction having neither prismatic nor aniseikonic elements.

It has been pointed out before that as a rule patients with meridional aniseikonic errors at oblique axes are not aware of difficulties in spatial orientation. Occasionally, however, such difficulties are reported. The following case is one in point.

CASE 7.—R. A. B., a man aged 24, assistant to a civil engineer, reported that he had worn glasses since childhood but that he had never seen well even with them. He complained of daily frontal and temporal headaches; burning, smarting and watering of the eyes; occasional vertical diplopia, and inability to read for more than half an hour. These difficulties prevented the intelligent young man from going to college. He also reported difficulties in judging distances; he was never good at sports which involved watching a ball and had never driven a car with confidence.

The patient was wearing a correction of — 1.00 D. sph. ⌒ — 2.50 D. cyl., axis 160 for the right eye and of — 1.00 D. sph. ⌒ — 3.50 D. cyl., axis 30 for the left eye.

Refraction and Visual Acuity.—The results of refraction were as follows:

Right eye: — 1.25 D. sph. ⌒ — 3.50 D. cyl., axis 170, vision 20/25 +

Left eye: — 1.00 D. sph. ⌒ — 4.00 D. cyl., axis 17, vision 20/30+

In view of the significant change, this refractive correction was prescribed. It helped the patient only so far as it gave him better visual acuity, but his symptoms persisted. One year later he returned for another examination. The results of refraction were unchanged. For distance (20 feet) with his correction there were 1 prism diopter of esophoria and 1 prism diopter of left hyperphoria. The left hyperphoria increased to 3 prism diopters in the right half of the field of fixation, an observation which aroused the suspicion that it was based on weakness of the left superior oblique muscle. This suspicion was strengthened by the patient's habit of tilting his head to the right shoulder and the presence, with the Volkmann disk, of a disclination of 5.5 degrees in the left eye (as opposed to a conclination of 1.6 degrees in the right eye). In stereoscopic examinations the patient could obtain, with difficulty, a score of 90 per cent on the graduated Keystone DB6 chart. The hyperphoria was thought to be a disturbing factor, and the patient was given, in addition to his refractive correction, prismatic fit-over lenses: ¾ prism diopter base up for the right eye and 1¼ prism diopters base down for the left eye. He was asked to wear these glasses for two months. On his return he reported that he had great

difficulty with these glasses and that they had had no beneficial effect on his symptoms. In addition to the trouble in judging distances, he now noticed spatial distortions. The floor appeared to rise toward him, and in motion picture theaters the projection screen appeared tipped toward him.

Space Eikonometer Readings. — Measurements for aniseikonia were as follows:

At axis 90: (left) 0.50 ± 0.25%
At axis 180: 0.00 ± 0.25%

The measured declination error, δ_m, varied within wide limits, from —1.38 to —1.10 degrees, and the patient had a low stereoscopic sensitivity. After several days of testing, the declination error became more certain and was estimated to be about —1.00 degree. The computed declination error, δ_c, was —1.50 degrees. Accordingly, the following equivalent aniseikonic correction was prescribed:

Right eye: 1.50% meridional magnification, axis 135
Left eye: 1.50% meridional magnification, axis 45

This correction was given the patient as fit-over size lenses to be worn with his refractive correction, incorporating prisms.

After the patient had worn these glasses two months, he returned for examination. Measurements on the space eikonometer with his aniseikonic fit-over lenses were as follows:

At axis 90: (left) 0.25% ± 0.25%
At axis 180: 0.00 ± 0.50%
Measured declination error, δ_m: 0.00 ± 0.40°

Without the fit-over lenses the measured declination error, δ_m, was —1.07° ± 0.38°. He reported that his symptoms were much improved, and he had done much more close work than in the past. Although he still had occasional headaches, they were less frequent and less severe than before. The distortions previously noticed had disappeared, and the patient found for the first time that he was able to follow the flight of a ball in a ball game. No change in the correction was made. The patient reported once more, three months later, when he stated that the improvement had continued. The measurements on the space eikonometer were essentially the same as before except for a slight increase in the measured declination error. The final prescription arrived at incorporated, aside from the refractive correction and the prisms, 2.00% meridional magnification, axis 135, for the right eye and 2.00% meridional magnification, axis 45, for the left eye.

It must be pointed out that the various factors involved in this case were corrected in steps and that the patient obtained relief only after the declination error had been corrected by oblique meridional size lenses. The vertical heterophoria was undoubtedly of importance in his visual problem. But that it was not the chief cause of the trouble is borne out by the patient's failure to obtain relief from the prisms alone. In fact, it may be assumed that the improvement in his binocular cooperation brought about by the prisms made the aniseikonic error all the more difficult to cope with. This is indicated by the increase in spatial distortions noticed by the patient while wearing the prisms. The existing cyclophoria may account for the fact that the declination error was not measured to the full, theoretically expected, amount.

In general, the difficulties encountered in the correction of astigmatic errors at oblique axes can be largely minimized by including an aniseikonic correction in the oblique axis. In some cases, as in the one which will be reported next, the patient is totally unable to wear the oblique refractive astigmatic correction for any length of time.

Case 8.—Mrs. E. W. P., a housewife aged 22, had ulcers of the cornea at the age of about 6 years and had had a great deal of trouble with her eyes. After only a few minutes of reading or sewing severe eyestrain, frontal headaches and nausea occurred. The patient was unable to watch more than half a moving picture without severe discomfort. She had had numerous refractive corrections, several in the two years prior to examination on the space eikonometer; she had also had an aniseikonic correction based on standard eikonometer measurements—all to no avail.

Refraction and Visual Acuity.—The results of refraction were as follows:

Right eye: + 1.25 D. sph. ⊂ — 0.50 D. cyl., axis 170, vision 20/20 +
Left eye: + 3.25 D. sph. ⊂ — 5.00 D. cyl., axis 25, vision 20/30 +

At 20 feet (6 meters) there were 1 to 2 prism diopters of exophoria and ½ to 1 prism diopter of right hyperphoria, and at 16 inches (40.6 cm.), 6 to 7 prism diopters of exophoria and ½ to 1 prism diopter of right hyperphoria. Examination for stereopsis showed fusion but no depth perception. Except for a fine superficial nubecula of the right cornea and some fine, old superficial maculas of the left cornea, both eyes were normal.

The patient could not tolerate the refractive correction for even a few minutes. Nausea and a pulling sensation in the eyes set in as soon as she put the glasses on. With one eye closed she had no such symptoms. Measurements on the space eikonometer were finally arrived at as follows:

Axis 90: (right) 2.25% ± 0.75%
Axis 180: (left) 0.50% to 1.00% ± 0.50%
Measured declination error, δ_m: —1.00° ± 0.30°
Computed declination error, δ_c: —1.59°

The patient was given overnight the following equivalent aniseikonic correction:

Right eye: 3.00% meridional magnification, axis 155
Left eye: 2.00% meridional magnification, axis 25

The correction was given in fit-over lenses for her refractive correction. The patient was able to wear this combination with reasonable comfort, and even to read for some time. In the course of the next two days repeated readings were made on the space eikonometer, with the result that the following correction was found which corrected the false appearances of the space eikonometer elements and allowed her to wear the glasses with so much comfort that she could read for two hours:

Right eye: + 1.25 D. sph. ⊂ — 0.50 D. cyl., axis 170 ⊂ 2.75% over-all magnification ⊂ 0.75% meridional magnification, axis 170.

Left eye: + 3.25 D. sph. ⊂ — 5.00 D. cyl., axis 25 ⊂ 4.00% meridional magnification, axis 45. The patient

has had these glasses for five months but was unable to come for reexamination, owing to an intercurrent illness.

Although a final judgment cannot be arrived at, since the patient has not been able to return for examination, this case shows that it is possible to enable a patient with an extreme astigmatic anisometropic refractive error to wear his glasses by correcting the declination error which is introduced by the spectacles. For theoretic reasons this is particularly interesting in cases in which the astigmatic error at the oblique axis is equal and symmetric in the two eyes.

Case 9.—Mrs. V. C. H., the wife of a farmer, was 49 when she was first seen at the Dartmouth Eye Institute. She complained of eyestrain, to which she had been subject since she was a child; burning of the eyes, and frontal headaches. She had worn glasses since the age of 20. In the past years the eyes had burned less and she had fewer headaches, but she was still unable to read for more than five minutes without discomfort.

During 1937, 1938 and 1939 she was given a number of refractive and aniseikonic corrections, without incorporation of corrections at oblique axes, none of which proved helpful. The last correction was as follows:

Right eye: + 5.00 D. sph. ⊃ — 1.75 D. cyl., axis 60
Left eye: + 4.75 D. sph. ⊃ — 1.75 D. cyl., axis 115 ⊃ 1.25% over-all magnification. Additional correction for near work of + 2.00 D. sph. was given for each eye.

The patient returned one and a half years later. At that time the refraction and visual acuity were as follows:

Right eye: + 5.50 D. sph. ⊃ — 1.75 D. cyl., axis 60, vision 20/20-2

Left eye: + 5.00 D. sph. ⊃ — 1.75 D. cyl., axis 120, vision 20/20-3

At 20 feet (6 meters) there was 5 to 6 prism diopters of esophoria, and at 16 inches (40.6 cm.), with an addition of + 2.00 D. sph. for each eye for reading, there was 2 to 3 prism diopters of exophoria. Examination for stereopsis revealed 100 per cent depth perception with the graduated Keystone DB6 chart.

Measurements on Space Eikonometer.—The measurements for aniseikonia were as follows:

At axis 90: (right) 0.50% ± 0.25%
At axis 180: (right) 0.50% ± 0.25%
Measured declination error, δ_m: + 0.85° ± 0.20°
Computed declination error, δ_c: + 1.70°

The patient was given the following equivalent aniseikonic correction in temporary form:

Right eye: + 5.50 D. sph. ⊃ — 1.75 D. cyl., axis 60 ⊃ 1.00% meridional magnification, axis 50

Left eye: + 5.00 D. sph. ⊃ — 1.75 D. cyl., axis 120 ⊃ 2.50% meridional magnification, axis 140.

For close work a + 2.00 sphere was added for each eye. The patient wore these glasses with great comfort for three months, after which time reexamination yielded essentially the same result as did the first examination, but the axes of the meridional size lenses were shifted to axis 60 in the right eye and to axis 150 in the left eye. The improvement continued, and the patient was given permanent glasses. Three months after receiving the permanent correction the patient was reexamined on the space eikonometer, and the declination error, δ_m, was found to be 0.00 ± 0.16 degree. She has worn the permanent glasses with great comfort for over a year.

We think that this case is of particular interest not only because it shows close agreement between the measured and the computed declination error and because good results were obtained with the oblique aniseikonic correction, but particularly because the refractive correction for this patient was entirely symmetric except for an insignificant amount of anisometropia in the spherical part.

As has been pointed out in the introduction of this paper, it is generally recognized that oblique astigmatic corrections often are not tolerated well by the patient. Various reasons have been given for the discomfort. Recently Sugar [25a] reported that many patients whose refraction is accurately determined in monocular vision are uncomfortable in binocular vision. In all 4 cases which Sugar reported a large oblique anisometropic astigmatism was present. He advocated the use of the "spherical equivalent," a technic which apparently makes for visual comfort at the expense of visual acuity. In commenting on Sugar's paper, O'Neill [26] and Lebensohn [27] pointed out that the anisometropic astigmatic corrections introduce a differential prismatic effect at the reading level, and they claimed that this is responsible for the patient's discomfort. Sugar,[28] in replying to these comments, stated that the prismatic differential effect of the glasses could not have played a part since the trial lenses were properly centered before the eyes and the test chart was 6 meters away. This statement, of course, is irrelevant to the problem.

The differential prismatic effect is undoubtedly a factor at the reading level, but we believe that the most important factor in the patient's comfort is the correction of the aniseikonic error produced by cylindric spectacle lenses. All the patients whom we have seen have had difficulties in distant vision, as well as in close work. Our contention is strengthened particularly by instances such as case 9, in which the spectacles contain symmetric lenses which do not introduce a differential prismatic effect at the reading level.

25a. Sugar, H. S.: Binocular Refraction with Cross Cylinder Technic, Arch. Ophth. **31**:34 (Jan.) 1944.

26. O'Neill, H.: Binocular Refraction with Cross Cylinder Technic, Correspondence, Arch. Ophth. **31**: 267 (March) 1944.

27. Lebensohn, J. E.: Binocular Refraction with Cross Cylinder Technic, Correspondence, Arch. Ophth. **31**:268 (March) 1944.

28. Sugar, H. S.: Binocular Refraction with Cross Cylinder Technic, Arch. Ophth. **31**:269 (March) 1944.

SUMMARY AND CONCLUSIONS

The discomfort experienced by patients who require an astigmatic correction at oblique axes appears to be a problem which is as old as modern refraction itself. Various explanations have been offered, both for the patient's asthenopic symptoms and for the spatial distortions caused by such corrections, but no means of relieving them effectively has been suggested in the past.

In this paper we have pointed out that the problem here is essentially the problem of aniseikonia, in particular one that involves a meridional aniseikonia at an oblique axis.

An aniseikonic correction at an oblique axis can be determined if, in addition to the usual measurements for the image size differences in the vertical and the horizontal meridian, a measurement of the declination error introduced by the aniseikonic error at the oblique axis is also obtained.

The term declination error designates a small angular deviation between the images in the two eyes of a vertical line in space. Such a deviation may be the result of the correction of astigmatism at an oblique axis, for this correction would introduce a meridional magnification or elongation of the dioptric image on the retina at an oblique axis. Associated with that magnification is a small rotary deviation of the images of vertical objects. This deviation, when existing between the images of the two eyes, would cause a particular stereoscopic spatial distortion. Under test conditions in which empiric factors to spatial localization are excluded, the declination errors can be measured by the spatial distortion associated with them. The declination error which might be expected to exist in the individual case on the basis of the astigmatic correction can be theoretically computed.

The space eikonometer is an instrument for measuring the declination error, as well as the image size differences in the horizontal and vertical meridians by means of the incorrect binocular spatial localization associated with the aniseikonia. From these measurements the aniseikonic correction involving a meridional correction at an oblique axis can be determined.

A study of the benefit which patients might derive from aniseikonic corrections at oblique axes was made. This paper presents the report on a first group of patients to whom such prescriptions were given. While many problems of a theoretic and practical nature remain to be solved, we feel justified in presenting the results obtained at this time.

Seventy-six patients were given prescriptions for aniseikonia at oblique axes on the basis of measurements on the space eikonometer. The data on 13 patients had to be discarded for lack of information or because of unusual complicating factors; of the remaining 63 patients, 36 were relieved of their symptoms by the correction to a notable degree; 16 were partially relieved, and 11 derived no benefit at all. This high percentage of positive results we consider significant, especially in view of the fact that the patient material was highly selected. The majority of these patients had asthenopic symptoms more severe than has the average patient. Others had ocular and extraocular factors that made a positive result often questionable, even prior to the examination.

Not for all patients did the measured declination error agree in amount and direction with that to be expected on the basis of the astigmatism. For many patients the measured declination error was smaller than the error theoretically expected; for others it was greater. Again, for others it was even in the opposite direction. Nevertheless, patients were helped by the correction of the measured declination error. Other patients had a spherical refractive correction or were even emmetropic, so that the computed declination error was zero. Notwithstanding this, these patients were rendered more comfortable by the correction based on the measurements.

For the time being there are two possible explanations for the measurements in such cases: Either the patients have an oblique basic aniseikonia which is not optical in origin, or they have a cyclophoria. Further research is required to solve this problem. A similar explanation must apply in the case of patients who have a large astigmatic error at oblique axes but for whom no declination error can be measured.

In several patients no relief was obtained by the correction, in spite of the fact that a definite declination error was measured and that it agreed well with what might be expected.

The correction of meridional aniseikonia at oblique axes is particularly helpful in cases in which the patient is absolutely unable to wear an oblique cylindric correction at oblique axes and yet his symptoms necessitate the wearing of such a correction.

The symptoms in these cases cannot be entirely due to a differential prismatic effect of the glasses. This is evident in cases of symmetric refractive errors in which the correction of the declination error afforded considerable relief. It

also follows from the cases in which relief was obtained by correction of the measured declination error which was the opposite of that which might be expected. In these cases the equivalent aniseikonic correction would tend to increase the differential prismatic effect of the glasses.

Mr. Leo F. Madigan assumed the responsibility of conducting the space eikonometer tests, and through his careful and patient approach to the work this study was made possible.

ABSTRACT OF DISCUSSION

DR. LAWRENCE T. POST, St. Louis: My studies on aniseikonia made personally in more than 450 patients and less personal on about 700 patients during the past ten years have been concerned with practice rather than theory. The figures which my associates and I have obtained indicate that about 80 per cent of patients for whom aniseikonic glasses were ordered definitely helped and that almost 50 per cent were made entirely comfortable. Replies were solicited only from persons who had worn their aniseikonic correction for at least nine months. The first 100 replies to the questionnaire were used for the report which was published in the *Southern Medical Journal* in July 1942, page 649. The first question which arises is whether the major factor in this increased comfort is the correction of aniseikonia or something else, such as a change in the refractive correction made at the time the eikonic lenses were given or the psychologic effect of the elaborate examination. It may also be asked whether the patient could have been relieved by some procedure other than the giving of the eikonic glasses, for example, orthoptic training. I have the impression that the eikonic correction is by far the most important factor in producing this increased comfort. It is not only possible but probable that some of these patients could have been helped by means other than aniseikonic lenses. I must add, however, that patients who, judging from our careful histories and ophthalmic examinations, probably had some additional pathologic condition, such as sinustis or an anomaly of the extraocular muscles, which caused the symptoms were referred for care of these conditions before being given eikonic glasses.

But I cannot refrain from asking, "What difference does it make whether the discomfort is primarily aniseikonic or muscular or psychic, if wearing of the eikonic glasses results in comfort? Why should one not as well give relief by the simple expedient of prescribing eikonic lenses as by an extensive course of orthoptic training?" To consider the more immediate problems presented by the authors, any one who has worked with aniseikonia has felt the need of an instrument that could measure this error. Perhaps ophthalmologists had not visualized the eikonometer as a possible means of giving comfort to the specific group of patients with astigmatism at oblique axes the correction of which did not produce comfort, so much as of helping patients with aniseikonia in an oblique plane which has a cause other than astigmatism. However, the application of the so-called space eikonometer in correction of declinations produced by astigmatism at oblique angles is obvious. The number of patients with such an error is probably relatively small. The ability of the eye to overcome muscular and refractive difficulties is amazing, as is the ability of the brain to tolerate deficiencies of muscles and dioptric systems. Thousands of people must have aniseikonia and uncorrected errors of refraction which do not inconvenience them. Probably increasing visual skills would help not only many patients who suffer from aniseikonia but many who have other ocular disabilities. Even granting this premise, most people do not have the time, the energy, the money or the inclination to train these skills, and for them, as well as for the great majority who could not be helped by such training, there will continue to be a need for refractive and eikonic corrections. The authors, in addition to presenting a beautiful theorem, have added a practical method of making a small group of patients comfortable. Aniseikonia can never be fully evaluated clinically or the eyes attain their maximum usefulness until two conditions have been fulfilled: first, the test must be made available to every ophthalmologist, and, second, the cost of eikonic lenses must not be prohibitive to the average patient.

DR. ERNEST A. W. SHEPPARD, Washington, D. C.: Early students of modern refraction and of ocular motility recognized deviations of the vertical meridians of the eyes. Stevens designated such a deviation as the declination error, and he and Savage emphasized its importance as a cause of asthenopia. The types of declination errors were not clearly understood, and therefore the treatment was unsatisfactory. The authors have described three types, on the basis of the etiologic factors: 1. Declination errors produced by cylindric lenses at oblique axes. In this type the degree of error is variable, being maximal when the meridian of the power of the lens is at 45 degrees or at 135 degrees. It is not amenable to correction surgically. It can be calculated mathematically, but the calculated amount does not always agree with the measured amount because of the influence of type 3. 2. Deviation errors associated with cyclophoria. In this type the degree of the declination error is the same in all meridians and therefore cannot be corrected by lenses. 3. Deviation errors associated with basic meridional aniseikonia. This type may exist with emmetropia or isometropia. It may influence the calculated amount in the first type, so that the measured amount may be less, may be zero or even of the opposite kind. If the declination error is sufficiently large to be recognized in the synoptophore, it should be made a

part of the orthoptic status. Certainly, ophthalmologists and orthoptic technicians should be familiar with the types. For diagnostic purposes it is more important to know the type than the degree. It is unfortunate that disturbances of ocular motility are more often treated according to degree than to type. Using the Ames space eikonometer, the authors have measured the declination error by the spatial distortion associated with it and have corrected it with iseikonic lenses. It is an important advance both in refraction and in ocular motility to have recognized the association of the two conditions. The results they have obtained are most gratifying. Most of the patients who come to the aniseikonia clinic have tried about everything to get relief. Many have suffered so long that they have acquired what is usually considered a neurosis. A careful history and examination will often reveal associated ocular, physical and psychologic factors. It is reasonable therefore that not all patients will be relieved by the iseikonic correction. The authors have recognized and correctly interpreted the fundamental problem of declination errors, classified them according to the etiologic factors, described an instrument for their measurement and reported results in the correction of such errors by iseikonic lenses. They recognize further problems presented by the solution of this one. Another aid has been added to the means of solution of the difficult problem of obstinate asthenopia.

Dr. Hermann M. Burian, Hanover, N. H.: I wish to thank Dr. Post and Dr. Sheppard for their thorough discussions. Dr. Post stated that a change in the refractive correction may be a source of relief for the patient. Of course, Professor Ogle and I have considered this. The table gives the pertinent information on the 63 patients on whom the study is based, including the refractive correction worn and the refractive correction prescribed. This will allow every one to judge for himself whether, and to what extent, this factor is of significance. The bearing on the results of the ability to overcome certain ocular difficulties, i. e., of the ability to adapt, is also discussed at length in the paper. I concur most heartily with Dr. Post's conclusions. I have maintained for years that if aniseikonia is as important as it is thought to be, its measurement should be so simplified as to enable any one to determine the aniseikonic error with the other ocular anomalies. This is not so simple as it sounds. Numerous difficulties had to be overcome. We have, however, in the past year succeeded in constructing a small instrument, based on the principle of the vectograph, which utilizes the test pattern of the space eikonometer. It is hoped that with this instrument, which should become available as soon as the present difficulties in obtaining materials are removed, the measurement of aniseikonia will be no more difficult than is, for example, the subjective determination of the axis and the amount of astigmatism. We expected to demonstrate a model at the present session, but this was not possible, for lack of space.

Dartmouth Eye Institute.

HETEROCHROMIA SYMPATHICA

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In a recent publication Hess [1] called attention to certain aspects of heterochromia sympathica in addition to the features usually described. He stressed the presence of status dysraphicus, which is evidence of involvement of the spinal cord and which may present the following clinical signs: (*a*) signs of localized muscular atrophy, (*b*) reflex abnormalities, (*c*) vasomotor changes, (*d*) disturbance in function of the urinary bladder and of the bowels, (*e*) abnormalities in the distribution of the fat of the body and (*f*) scoliosis. In accordance with von Herrenschwand's suggestion,[2] heterochromia sympathica became recognized as a separate clinical entity. Extraocular involvement of the sympathetic nervous system in this condition is not unknown. Mayou [3] recorded a case of heterochromia sympathica following birth injury to the sympathetic system. Waardenburg,[4] Metzner and Wölfflin [5] and Bistis [6] described involvement of the facial nerve and cervical portion of the sympathetic trunk (Horner's syndrome and hemiatrophy) in association with heterochromia sympathica. Cases have been recorded in which injury of the cervical portion of the sympathetic trunk by trauma [7] or at operation [8] was followed by heterochromia. This occurred in young persons below the age of 25 years. Passow [9] first associated heterochromia with skeletal and cranial structural anomalies.

There is enough evidence to show that with a certain type of heterochromia [10] the sympathetic nervous system is involved. Four cases which may be of interest are here described. Because we have seen so few cases of heterochromia sympathica (4 in a series of 13,450 cases) and because there was evidence of birth injury in 2 of them, we consider our cases worthy of record. It is noteworthy that among the 13,450 cases observed by us in South Africa, not 1 instance of heterochromic cyclitis was encountered. Seventeen cases (in the series of 13,450 cases) of simple heterochromia are on our records. Although we observed between 3,000 and 4,000 South African Negroes, not 1 case of heterochromia simplex or heterochromia complicata was seen.

Of our 4 cases of heterochromia sympathica the condition followed birth injury in 2; sympathetic disturbances became evident soon after birth in the third, and changes in color of the iris occurred during the patient's lifetime in the fourth.

REPORT OF CASES

CASE 1.—T. W. B., a man aged 31, stated that he was born "with his head twisted on to his left shoulder." Delivery was by forceps extraction. He had an oblique linear scar on the right side of his forehead and another over the right parietal region of his scalp. The iris of the right eye was brown, and that of the left eye was blue with a few brown specks in it. The right palpebral fissure was 2 mm. wider than the left. The diameter of the right pupil was 3.5 mm., and that of the left pupil, 2.5 mm. The skin of the left side of the forehead and the eyelids was continually wet with perspiration throughout both summer and winter. No perceptible differences were to be seen in the fundi. Refractive errors required the following correction: right eye, —0.5 D. sph. ⌒ —0.5 D. cyl., axis 180; left eye, —0.5 D. sph. ⌒ —1.5 D. cyl., axis 180. With correction visual acuity was 6/6 in each eye.

CASE 2.—J. H. D., a sergeant aged 38, stated that his was an "instrument birth" from which he retained a depression in the bone on the left side of his forehead and a scar over the right side of his lower jaw opposite the second premolar tooth. The color of the right iris was blue, and that of the left iris, light brown. The right palpebral fissure was narrower than the left. The diameter of the right pupil was 2.5 mm., and that of the left pupil, 3 mm. The fundi did not differ from each other. Refraction showed rather mild simple hyperopic astigmatism with horizontal axes in each eye. No abnormality in function of the skin was seen.

In both these cases the physician reported that no other signs of sympathetic involvement

1. Hess, L.: Heterochromia of Iris, Arch. Ophth. **30**:93 (July) 1943.
2. von Herrenschwand, F.: Klin. Monatsbl. f. Augenh. **60**:467, 1918.
3. Mayou, M. S.: Tr. Ophth. Soc. U. Kingdom **36**: 410, 1916.
4. Waardenburg, P. J.: Nederl. tijdschr. v. geneesk. **1**:22, 1929.
5. Metzner and Wölfflin: Arch. f. Ophth. **91**:167, 1916.
6. Bistis, J.: Ztschr. f. Augenh. **82**:180, 1934.
7. Lazarescu, D., and Lazarescu, E.: Ann. d'ocul. **170**:767, 1933.
8. Bistis, J.: Ann. d'ocul. **45**:569, 1928.
9. Passow, A.: Arch. f. Augenh. **108**:137, 1933.
10. W. S. Duke-Elder (Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 1403) classifies heterochromia thus: (1) simple heterochromia; (2) complicated heterochromia, (*a*) heterochromia cyclitis and (*b*) sympathetic heterochromia.

existed. There was no family history of heterochromia. In these 2 cases the cervical sympathetic fibers must have been damaged at birth, with the result that the disparity in color of the iris developed.

CASE 3.—S. C. van W., a boy 6 years of age, had a normal birth and infancy. There was nothing of importance in the family history. From infancy the left side of his face down to the chin became flushed at times of exertion or emotion The cornea of the left eye was 0.5 mm. larger than that of the right eye in both the vertical and the horizontal diameter. The left pupil was larger than the right. The iris of the left eye was brown, and that of the right eye was blue. The palpebral fissure of the left eye was wider than that of the right eye. No abnormality in function of the sweat glands or in general constitution could be found by the pediatrician. With a + 0.75 D. sphere for the right eye vision was 6/6 in that eye, and with an 8 D. sphere for the left eye vision was barely 6/60 in that eye. The left eye was congenitally amblyopic.

In this case no skeletal differences existed, but there was obviously an imbalance of sympathetic function on the two sides of the body, probably of congenital origin.

CASE 4.—H. W., a man aged 45, had an interesting history of changes in the color of his irises. His family history revealed no clues to account for this condition. He stated that his health had always been exceptionally good, but he had had thorough internal medical and roentgenologic examinations for the presence of any possible condition, such as unilateral silicosis or tuberculosis,[11] which could have caused a disturbance of the sympathetic nervous system. None had been found. His complexion for a European born in England, and in spite of the fact that he was an underground worker in a South African gold mine, was singularly ashen brown. I examined his old military papers, the first of which, dated June 24, 1916, stated: "Color of eyes, light gray." He offered the information that a short time after this record was made his friends told him that the color of his eyes had changed. He himself had noticed it. The second certificate, dated Oct. 12, 1918, stated: "Color of eyes, brown." I saw him in January 1942, when the right eye was blue and the left eye was brown. No other differences were noted between the two eyes. He stated that the latest change in color of the irises (heterochromia) had taken place gradually during the last fourteen years.

We are at a loss to explain the mechanism in case 4, but we regard the condition as heterochromia sympathica because of the changes which the color of the irises seemed to have undergone during the patient's lifetime. There is, of course, the possibility that the earlier examinations of the eyes were made in a haphazard manner, that is, only one eye may have been looked at on each occasion. However, such changes in color of the iris may occur, and the recording of this case may bring forth descriptions of other cases of similar nature in which other causal factors exist.

6 and 7 Mariners, Jeppe and Joubert Streets.

11. Giamantoni and Possente: Ann. di ottal. e clin. ocul. **61**:823, 1933; cited by Duke-Elder.[10]

Clinical Notes

PLASTIC CORNEAL BATH FOR APPLICATION OF PENICILLIN

CAPTAIN HUGH A. G. DUNCAN, ROYAL CANADIAN ARMY MEDICAL CORPS

Recent work by von Sallmann and Meyer [1] has indicated that penicillin is of little value in the treatment of intraocular infections if given intramuscularly or instilled as drops. Their results suggested that iontophoresis and the use of a corneal bath plus a wetting agent are the only satisfactory means of introducing a sufficient concentration of penicillin into the anterior chamber. According to their report, iontophoresis is more effective, but with the corneal bath an adequate concentration can be obtained. The latter method is, moreover, much easier to carry out, especially for ophthalmologists in service.

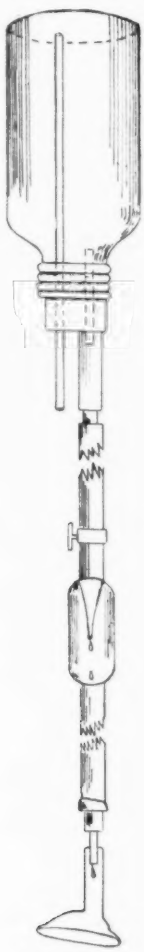

Fig. 1.—Plastic corneal bath.

A simple corneal bath of plastic (acrylic acid derivative) has been devised, with the technical help of Major J. M. McDougall, Canadian Dental Corps, and his assistant, Sgt. H. Travers. It is modeled from an average-sized contact lens. The corneal curvature is made greater than that of an ordinary contact lens. A plastic tube is molded in place between the apex and the scleral gutter at an angle of 20 degrees with the anteroposterior axis. The tube is placed below the apex in order that when the lids are closed and the eye turns up the dome may not be displaced from the cornea. The angle of the tube is such that when the patient is on his back the tube is vertical; thus, with the drip method, to be

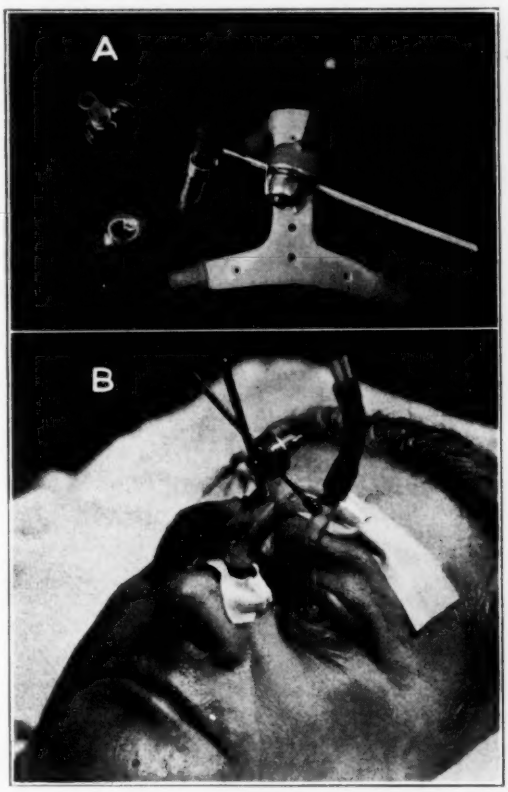

Fig. 2.—Photographs of the plastic corneal bath.

From the Canadian General Hospital no. 4.

1. von Sallmann, L., and Meyer, K.: Penetration of Penicillin into Eye, Arch. Ophth. **31**:1-7 (Jan.) 1944.

described, the tubing does not drag on the plastic inlet to the bath (fig. 2).

If a constant bath is desired, a drip can be arranged from an ordinary set for continuous intravenous infusion, with the tubing suspended vertically. No weight is placed on the bath. A gutta-percha ring can be placed around the tubing above its outlet, to prevent the tip from striking the cornea if the tubing should fall accidentally. With many types of sets for intravenous infusion this adaptation is unnecessary.

For this bath, as well as for the drops, I have used penicillin in a strength of 1,000 units per cubic centimeter, rather than the more common concentration of 500 units per cubic centimeter, because experimentally it has been shown that the stronger concentration remains effective at ward temperature for three to four days. The weaker concentration remains effective for less than half that time.

In using the apparatus described here, one must be careful not to leave it in a badly inflamed eye too long because of the danger of pressure necrosis. The patients with whom I have used this bath bore it with comparative comfort. Nupercaine hydrochloride was added to the penicillin, as suggested by von Sallmann and Meyer.

I have not yet had sufficient experience to warrant definite conclusions as to the optimum frequency or duration of the treatments. However, from clinical experience with iontophoresis of penicillin, I believe that daily baths for comparatively short periods may be satisfactory, especially when they are supplemented with intramuscular administration of penicillin.

This article is published at the suggestion of Lt. Col. R. E. Wright, Indian Medical Service, who gave valuable advice; it is felt that a suitable corneal bath may be of material aid in the treatment of war injuries with resultant intraocular infection.

Correspondence

BILATERAL INFANTILE GLAUCOMA ASSOCIATED WITH BILATERAL CONGENITAL HEMANGIOMA

To the Editor:—A recently recorded case of "bilateral infantile glaucoma associated with bilateral hemangioma congenital (nevus flammeus)" (ARCH. OPHTH. **32:** 214 [Sept.] 1944) has come to my attention. The author, A. M. Rodigina, states that a review of the literature failed to reveal any case of a similar condition. I should like, therefore, to suggest that the following cases of similar nature have been reported and listed by J. Ringland Anderson, in his monograph entitled "Hydrophthalmia or Congenital Glaucoma" (London, Cambridge University Press, 1939). These cases are listed as instances of bilateral hydrophthalmos and bilateral facial nevi (Beltman, J.: *Arch. f. Ophth.* **59:** 502, 1904; Marchesani, O.: *Wien. med. Wchnschr.* **75:** 2538, 1925; Horay, G.: *Zentralbl. f. d. ges. Ophth.* **20:** 793, 1929; Dunphy, E. B.: *Am. J. Ophth.* **18:** 714, 1935; Perera, C. A.: Bilateral Buphthalmos Associated with Naevus Flammeus, ARCH. OPHTH. **14:** 626 [Oct.] 1935; Fleischer, B.: *Klin. Monatsbl. f. Augenh.* **61:** 152, 1918).

It is presumed that by the term infantile glaucoma the author means a primary type of congenital glaucoma, or hydrophthalmos, which according to Anderson's definition would include the case under discussion. Besides these cases listed by Anderson, 4 cases in which the condition was considered as "bilateral glaucoma (other than congenital) with bilateral facial nevi" have been recorded, and in all these cases glaucoma had developed before the age of 20 years. They are: Biro's case, with onset at 8 years; Knapp's case, with onset at 4 years (mild hydrophthalmos?); Horay's case, with blindness at 19 years, and Mehney's case, with blindness at 20 years. Since the condition in the author's case appeared between the ages of 9 and 11 years and since the diameter of the right cornea was 13 mm. and that of the left cornea was 11 mm., it appears that hydrophthalmos was present in the right eye, but probably not in the left eye. Accordingly, in the sense intended in the report, the glaucoma in the left eye was probably not of the true congenital type; so other cases of this type, in which bilateral facial nevus coexists with secondary glaucoma, or the pseudoglaucoma of Ballantyne (*Brit. J. Ophth.* **14:** 481, 1930; **24:** 65, 1940), may also be noted.

It may be mentioned at this time that of 60 cases of hydrophthalmos associated with facial nevi, 35 per cent showed bilateral nevus. Again, it may be interesting to point out that in Anderson's series, the hydrophthalmos was bilateral in 85 per cent of cases. From this one would assume that if the patient had bilateral nevus, he would be more than likely to have bilateral glaucoma. Yet in all cases of hydrophthalmos with facial nevi (60 cases) bilateral glaucoma was present in only 13 per cent. Does this indicate that the cases have not been followed to a conclusion, or does it suggest an element of prognosis? Is the relation of facial nevus to hydrophthalmos of no significance? May not the facial nevus be related to a glaucoma of delayed development, which occasionally appears as hydrophthalmos very early in one eye and as another type of glaucoma later in the second eye, modified in character by changes occurring during the growth and development of the eye?

The author states that "roentgenograms of the skull indicated no pathologic change in the sella turcica." I believe there have been reported only 3 or 4 cases of this condition in which changes were observed in the pituitary gland, and in these cases no relationship between the angioma and the changes in the pituitary was apparent. It would seem to be more important to make roentgenograms of the skull for the presence of calcification, particularly of the occiput, for angiomas of the meninges, cortex and cerebellum may be present. The relationship between cutaneous nevus in the distribution of the trigeminal nerve and angioma of the occiput, both meningeal and cortical, points to an embryonic anlage. It has been shown that the roentgenographic report for calcification will be negative if the tumor is present but has not as yet become calcified. Anderson has pointed out that glaucoma develops only in eyes with tarsal and conjunctival angiomatosis, and not in eyes in which these appendages are not involved.

EMANUEL ROSEN, CAPT., M.C., A.U.S.

RETINAL CHANGES ASSOCIATED WITH DIABETES AND WITH HYPERTENSION

To the Editor:—In his article, "Retinal Changes Associated with Diabetes and with Hypertension," published in the February 1945 issue of the ARCHIVES, Ballantyne presents illustrations showing dilatations of capillaries in the retina which he calls microaneurysms. He states that in fixed preparations "the aneurysms can be clearly seen in greater number than on oph-

thalmoscopic examination." I do not know whether the term aneurysm can properly be applied to a capillary dilatation, but from his illustrations and description there is no doubt that Ballantyne saw numerous capillary dilatations. This is in accord with the assumption which I made in my publication "Problem of Diabetic Retinitis" (ARCH. OPHTH. **25:** 139-148 [Jan.] 1941). I expressed the opinion that in view of the present understanding of local circulatory disturbances in an organ, "the hemorrhages in the retina must be considered as the result of prestasis, that is, the result of slowing of the blood flow through dilated terminal vessel units" (page 143). Ballantyne's capillary microaneurysms seem to be nothing more than irregularly dilated capillaries presenting a condition of prestasis. Undoubtedly, as Ballantyne says, the dilatations are "the earliest manifestation of diabetic retinopathy" and "should throw some light on the pathogenesis of that condition."

There is, however, no necessity of assuming, as Ballantyne does, that unknown toxic factors are responsible for either the capillary dilatations or the occurrence of the hemorrhages. (Any comparison of human diabetes with diabetes experimentally induced with alloxan should not, of course, be made; the two conditions have nothing in common.) Hemorrhages as a result of diapedesis from capillaries occur whenever and wherever there are dilated capillaries with a condition of prestasis. This is a "normal" pathologic event. Also, in cases of diabetes, even of long standing, especially of the milder form, there are no known toxic factors. The only abnormality in such cases is the hyperglycemia, and this it is, as I have said in my article, which stands in a casual relation to diabetic retinopathy and is, in all probability, responsible for the capillary dilatations, or Ballantyne's "microaneurysms."

The changes in the retinal veins mentioned by Ballantyne occur to a notable degree only in a comparatively small number of cases of diabetic retinopathy. Together with formation of new vessels, they characterize the peculiar diabetic retinitis proliferans, the histologic features of which have been described by Klien. Only recently I was shown a case in which the fundus of the left eye was literally filled with a mass of newly formed vessels, while in the fundus of the right eye there could be seen the beginning of such vessel formation, together with hemorrhages and all manner of changes in the veins. The pathogenesis of these changes is more difficult to explain.

Finally, I think, one should not compare the changes in the fundus occurring in cases of arterial hypertension with those associated with diabetic retinopathy. Fundamentally, the alterations have nothing in common. The statement that in diabetes the "changes are primarily in the capillaries and venules, while in the hypertensive type they are in the arterioles and precapillaries" is not acceptable.

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239 Central Park West.

ILLUMINATION FOR OPHTHALMOLOGISTS

To the Editor:—In the January 1945 issue of the ARCHIVES, Dr. Legrand H. Hardy and Dr. Gertrude Rand present a paper entitled "Elementary Illumination for the Ophthalmologist." The scientific specification of foot candles, or intensity of illumination, has been one of the major objectives of researches in seeing for many years, and new knowledge and technics have crystallized in the past decade. The authors treat this important aspect meagerly. In fact, any one familiar with the growth of knowledge pertaining to the relations of illumination to the visibility of visual tasks and the complex relationships of light, vision and seeing knows that much of the progress in this direction has been made since 1931. The authors ignore this fact and quote Troland as of 1931 with a finality that is ridiculous. They might as well ignore the "miracle drugs" which have come into medical practice since that time and all the great progress in medicine, surgery and other sciences and practices of the past decade. They even cite Katz's statement, made in 1896! This was certainly the period of antiquity in lighting knowledge and practice, and it approaches a similar period in some major aspects of medical, ophthalmologic and other practices which involve human welfare.

To any one sincerely interested in knowledge of the relationships of light and sight and of the relations of these phenomena to visibility in an extreme variety of visual tasks, many scientific papers are available in appropriate journals published since 1931. The work of my associates and me, which the authors mention casually in passing, is ignored, together with all the work of other investigators. The authors should know that our researches in seeing are prosecuted with the same freedom and desire to ascertain the truth as exist in all General Electric laboratories, whose accomplishments speak for themselves. Why do the authors ignore the results of extensive researches of the past thirteen years which are revolutionizing lighting practice and are providing for the first time a scientific foundation for the specification of light and lighting? There may be an answer to this question, but it can scarcely be an excuse for the amateurish and antiquated discussion of the important matter of illumination. Much of the revolutionary knowledge and some of the new criteria, technics and devices have interested many ophthalmologists, who recognize that seeing involves other sciences and practices which affect the efficiency, safety and welfare of human beings throughout the present day artificial world of prolonged critical

seeing. Certainly, human eyes and human seeing machines are entitled to all the help that is available. They will not get it from persons who assume that the growth of knowledge ceased in 1931.

MATTHEW LUCKIESH.

Lighting Research Laboratory, General Electric Company, Nela Park, Cleveland.

To the Editor:—As Mr. Luckiesh has paid no attention to either our title or our references to recent and reliable sources of information on illumination he does not move us to change any statement we have made.

This note gives us the opportunity of correcting a typographic error on page 2 where a reciprocal sign was omitted. There are $\frac{1000}{1.076} = 929$ sq. cm. in a square foot, hence the foot lambert, as stated, equals 1.076 millilamberts.

LEGRAND H. HARDY, M.D.
AND
GERTRUDE RAND, Ph.D.

23 East Seventy-Ninth Street, New York.

INCOMPATIBILITY OF SILVER NITRATE AND SULFATHIAZOLE

To the Editor:—Since the publication of an article by D. F. Gillette entitled "A New Aid in Removal of Foreign Bodies of the Cornea" (ARCH. OPHTH. **31:** 129-130 [Feb.] 1944), my associates and I have been applying a 1 per cent solution of silver nitrate to the site of a foreign body. We agree with the author that the procedure facilitates the removal of metallic foreign bodies embedded in the epithelium.

In 2 of approximately 100 patients so treated a dense, grayish white precipitate has developed in or below Bowman's membrane about the site of the foreign body on application of the silver. In the first patient the area was not in the visual axis. Treatment was conservative, and the lesion epithelized satisfactorily. During the six months in which the precipitate has been under observation it has faded slightly, but is still conspicuous. Since we had used no other medication except tetracaine hydrochloride and the patient was not taking drugs, we could not explain a similar precipitate which appeared on application of silver nitrate to the cornea of another patient. Two days later, when it was evident that a conspicuous tattoo had been produced, the area was delineated, the cornea split superficially and the precipitate excised. The area healed smoothly, and normal visual acuity was obtained in the eye.

A history was obtained of the previous instillation of sulfathiazole ointment in this eye, as well as in other eyes with embedded foreign bodies which were subsequently treated with silver.

The precipitate dissolved readily in nitric acid and showed a strong reaction to silver. Unfortunately, there was insufficient quantity for further tests.

When 1 per cent silver nitrate is dropped into a saturated solution of sulfathiazole a fine white precipitate forms. Unfortunately, I have not been able to discover the exact nature of this precipitate. However, in a personal communication, Dr. Charles Allen Thomas, central research director of the Monsanto Chemical Company, suggests that it is either silver sulfathiazole or the silver salt of aminothiazole.

Partial hydrolysis to aminothiazole or metathesis, with the formation of sodium sulfathiazole, may form intermediate reactions which determine the extent of the precipitation. However, for practical purposes, in the absence of a more specific explanation of the phenomenon, one may assume that a break in Bowman's membrane allows a significant localized absorption of sulfathiazole and the formation of an insoluble precipitate on contact with silver nitrate. In cases in which no break in Bowman's membrane occurs, one may assume that there is too little absorption of the sulfathiazole to produce the insoluble precipitate.

Caution should be used in the application of silver nitrate to a cornea in which Bowman's membrane has been broken and which has been exposed to sulfathiazole.

COMMANDER A. M. CULLER (MC), U.S.N.R.

DEFECTS IN THE VISUAL FIELD PRODUCED BY HYALINE BODIES IN THE OPTIC DISKS

To the Editor:—In his interesting article entitled "Defects in Visual Fields Produced by Hyaline Bodies in the Optic Disks" (ARCH. OPHTH. **32:** 56 [July] 1944), Dr. C. Wilbur Rucker presents the changes in the visual fields in 5 cases of hyaline bodies in the optic disk. The fields presented were remarkably similar to each other and to the fields characteristic of glaucoma simplex. Since the author has not drawn attention to this fact, I believe it should be pointed out. In 4 of the 5 cases there were defects in the lower nasal fields, and in 2 cases, Seidel scotomas; examination in case 5 revealed a nasal step in each eye; in case 4, baring of the blind-spots, and in case 3, loss of the nasal fields and ring scotomas. If the field defects were analyzed for the internal isopters by test objects of smaller angles, viz., 1/1,000, 1/2,000, 1/4,000, I believe one would see (1) the ring scotoma ending in a nasal step, (2) a break-through of the Seidel signs to the nasal periphery and (3) additional nasal steps.

The author has presented the fields without drawing any conclusion. I think it should be pointed out that the fields presented are not "the usual varieties of defects in the visual fields caused by hyaline bodies in the optic disks," but are defects produced by any lesion at the nerve head. The fields presented uphold the generalization that a lesion at the optic disk produces peripheral field defects pointing to the disk and are due to lesions in the nerve bundles

at the disk. Other examples of peripheral field defects produced by a lesion of the disk are glaucoma, choroiditis juxtapapillaris of Jensen, papilledema and arterial occlusion.

The article has bearing on the various views as to the relation of the peripheral and the central retinal fibers as they pass into the papilla. According to Wilbrand and Saenger, and van der Hoeve, the fibers from the periphery of the retina pass to the outside of the nerve, and the peripapillary fibers, to its center. Since the physiologic cup exposes the most central fibers of the disk to pathologic change and since a pathologic process at the disk gives peripheral defects, it is likely that the views of the aforementioned authors are not correct.

I believe Dr. Rucker has presented additional clinical evidence in support of the view that the most central fibers at the disk represent the retinal periphery.

JACK V. LISMAN, M.D., New York.
654 Madison Avenue.

To the Editor:—I doubt whether my brief report of visual field defects produced by hyaline bodies in the optic disk contributes anything to knowledge of the arrangement of the nerve fibers in the disk. The hyaline masses are usually so irregularly distributed, and sometimes so deeply buried, that one cannot be certain as to which bundles of nerve fibers they interrupt.

I appreciate the interest of Dr. Lisman in this study.

C. W. RUCKER, M.D., Rochester, Minn.
The Mayo Clinic.

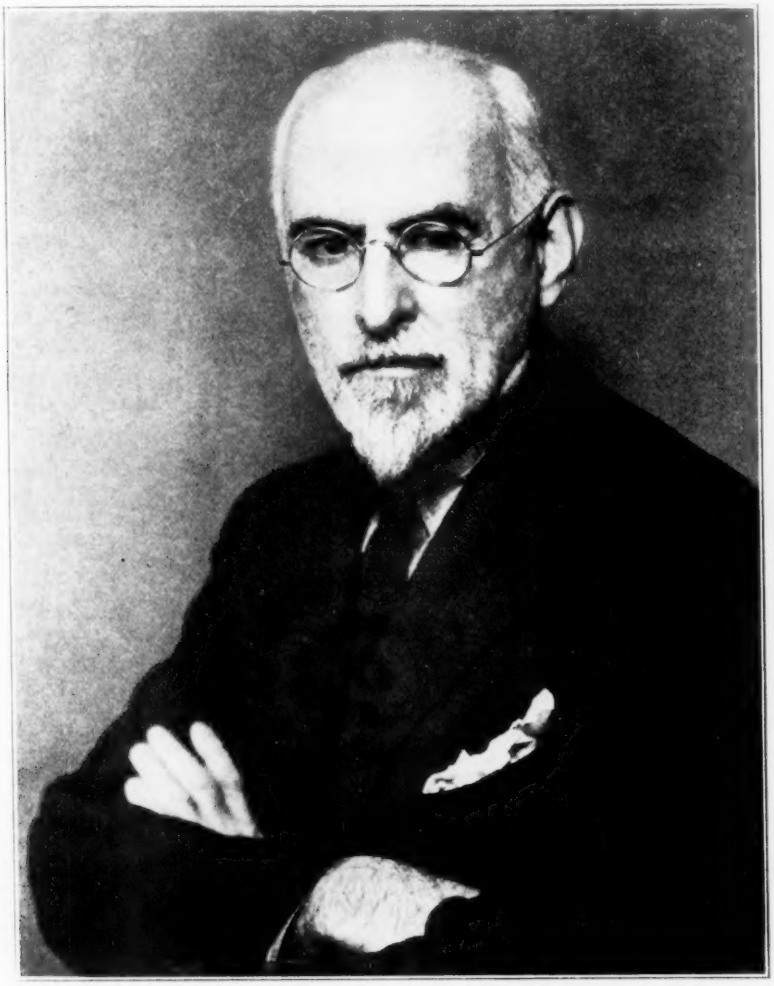
Obituaries

ELLICE M. ALGER, M.D.

1870 - 1945

Dr. Ellice M. Alger was born in Burlington, Vt., in 1870, and died at the New York Post-Graduate Hospital on Feb. 18, 1945, after an illness of several months. He is survived by his widow, Mrs. Louise Stevenson Alger, and a brother, George S. Alger.

He had been a member of the American Ophthalmological Society since 1915 and was also a fellow of the American College of Surgeons and a member of the New York Academy of Medicine. He was former chairman of the section of ophthalmology of the Academy, as



ELLICE M. ALGER, M.D.
1870 - 1945

Dr. Alger was graduated from the University of Vermont in 1890 and from its medical school three years later. He became instructor of ophthalmology at the New York Post-Graduate Hospital in 1902, receiving his professorship in 1912 and retiring in 1938.

well as a former president of the New York Ophthalmological Society.

In his fifty years of ophthalmic practice, Dr. Alger published several important papers. During his later years he became deeply interested in conditions relating to industrial oph-

thalmology. He was a pioneer in the campaign to improve industrial lighting and in instituting safeguards against ocular hazards in industry. In 1915 Dr. Alger assisted in the organization of the National Society for the Prevention of Blindness, serving continuously on its board of directors. Previously he was a member of the New York Association for the Blind.

In 1936 Dr. Alger was awarded the Leslie Dana gold medal for his outstanding achievements in the prevention of blindness and the conservation of vision. This medal is presented annually by the Research Association of Ophthalmology.

Dr. Alger's kindness and sympathy to clinic patients was significant of his character. His students were stimulated in the study of ophthalmology and profited by his practical demonstrations. His colleagues admired him for his modesty, friendliness and sincerity.

MARTIN COHEN.

THOMAS HARRISON BUTLER, D.M. (OXFORD), F.R.C.S.
1871-1945

Thomas Harrison Butler, the eminent English ophthalmologist, died suddenly at Hampton-in-Arden, Warwickshire, England, on Jan. 29, 1945. He was born at Stanhope, County Durham, England, on March 18, 1871. As the recipient of a scholarship, he entered Corpus Christi College, Oxford, in 1889. Here he won first class honors in natural science, graduating with the degree of Bachelor of Arts in 1893. He qualified from St. Bartholomew's Hospital in 1895. The year following he was awarded the Radcliffe Traveling Fellowship of the University of Oxford, which made it possible for him to spend several years in work in the great eye clinics on the Continent. For a time he was plague medical officer at Durban and harbor physician at Port Elizabeth, South Africa. The experience thus gained enabled him to write a thesis on the subject of plague, which won for him his doctorate at Oxford in 1902. For the next four years he served as assistant surgeon to the St. John Ophthalmic Hospital in Jerusalem.

On his return to England from Jerusalem, Dr. Butler wrote the British abstracts for these ARCHIVES— a task that he carried on for many years. He contributed a number of original articles to this publication, beginning with a paper on the treatment of trichiasis, in 1908.

Dr. Butler settled down to the practice of ophthalmology in Coventry and Leamington. His associations with these two cities covered a period of over forty years. His reputation spread far beyond the Midlands, until he became known to the medical profession throughout the English-speaking world.

In 1925 he attended Professor Vogt's course on use of the slit lamp, in Zurich. Two years later he published his book, entitled, "An Illustrated Guide to the Slit Lamp." This was the first book on the subject to be written in the English language, and it is as its author that Dr. Butler is best known. Being a gifted draftsman, he was able to provide the numerous illustrations in his book himself, and this he did for all his articles, which were many and mostly short.

Dr. Butler came to occupy high places in his specialty. To mention by no means all, he was at various times president of the Ophthalmological Society of the United Kingdom, of the ophthalmic section of the Royal Society of Medicine and of the ophthalmic section of the British Medical Association. He had the distinction of being selected to deliver the Doyne Memorial Lecture and the Montgomery Lectures. He was a most active member of the Oxford Ophthalmological Congress from the time of its foundation, serving on its council for many years. He will be sorely missed at its meetings, especially by those who came from great distances. It was his habit to single them out and take an interest in them. In recognition of his talents and energy and of the good use that he made of them, the Royal College of Surgeons elected him to the honorary fellowship in 1941.

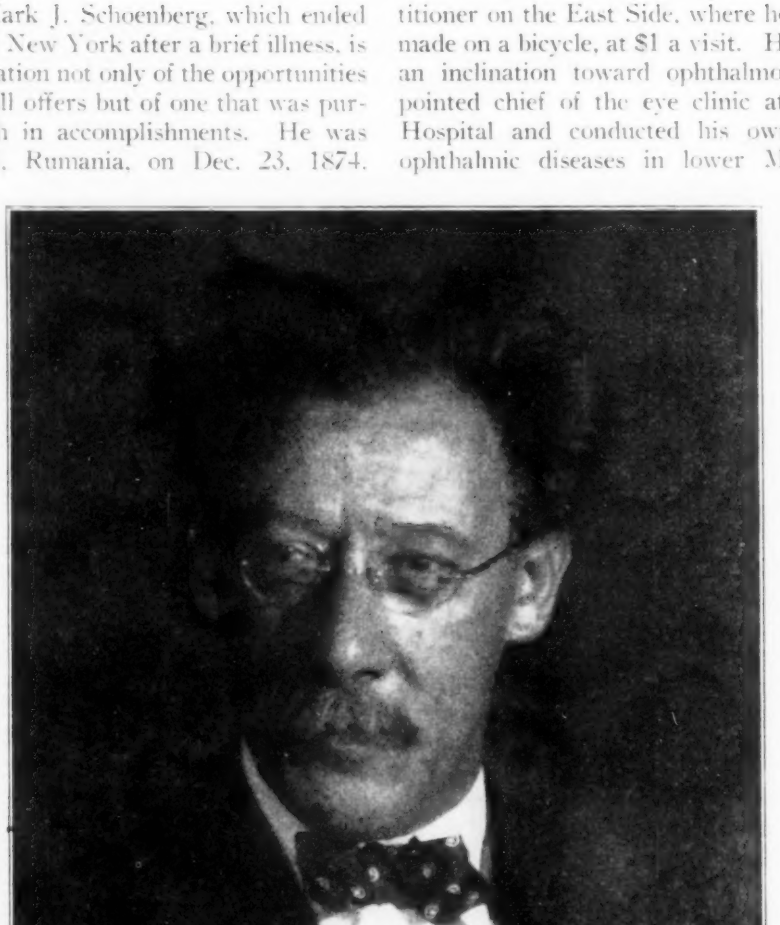
No sketch of Dr. Butler's career would be adequate without mentioning the fact that in the yachting world he was widely known as a designer of small cruising yachts. This was his hobby.

He died in harness. Owing to the exigency of the times, he returned from retirement to take up clinical work in Birmingham, in an endeavor to replace a son who had been called to the armed forces.

BERNARD SAMUELS.

MARK J. SCHOENBERG, M.D.
1874-1945

The life of Mark J. Schoenberg, which ended Feb. 15, 1945 in New York after a brief illness, is a brilliant illustration not only of the opportunities that America still offers but of one that was purposeful and rich in accomplishments. He was born in Pitesci, Rumania, on Dec. 23, 1874.



MARK J. SCHOENBERG, M.D.
1874-1945

Seeing the difficulty of his coreligionists in obtaining admission to the city high school, he organized and became a teacher in a new one with more democratic privileges. This training helped him in his later, lucid medical writings and teachings. He graduated from the Bucharest Medical University in 1898 and came to this country in 1900. He was first a general practitioner on the East Side, where house calls were made on a bicycle, at $1 a visit. He soon showed an inclination toward ophthalmology, was appointed chief of the eye clinic at Mount Sinai Hospital and conducted his own hospital for ophthalmic diseases in lower Manhattan. In 1908 he became connected with the Herman Knapp Memorial Eye Hospital and acted as attending surgeon from 1918 to 1935, when he retired. However, he continued his interest in the glaucoma clinic which he had started in that hospital, as the first in New York. His loyalty to the interests of this institution was outstanding, and he was especially helpful in the all day, six

month postgraduate course which were given for several years after 1918. He was instructor in ophthalmology at Columbia University College of Physicians and Surgeons. In 1919 he was made a member of the American Ophthalmological Society, his thesis being entitled "Remarks on Diagnosis and Treatment of Luetic Involvement of the Optic Pathways." He was also a member of the American Academy of Ophthalmology and Otolaryngology and the New York Academy of Medicine and served as chairman of the Section of Ophthalmology of the Academy in 1932 and 1933. He was the founder of the New York Society for Clinical Ophthalmology and acted as its first president, in 1935. He was consulting ophthalmologist to the Bronx Hospital and to the Manhattan Eye, Ear and Throat Hospital, where he was director of the glaucoma clinic. He was chairman of the committee for glaucoma of the National Society for the Prevention of Blindness.

Dr. Schoenberg was both loved and respected, loved for his kindness to every one, even those with whom he disagreed, and respected for his scientific accomplishments. He was by nature sensitive, a trait which enabled him to detect early expressions of disease in his patients. He was modest and enthusiastic, in all a highly successful practitioner, always thirsting for knowledge. He labored long before presenting his conclusions, and then gave only facts that were beyond refutation. He emphasized careful, complete history taking in every case, including information on diet and possible psychosomatic relations. He never lost the common touch. His humble and difficult beginning always tied his sympathies with the special problems of the poor patient. He encouraged writing by his assistants, not only by personal help but with monetary rewards. He had a most stimulating curiosity concerning medical facts, he tried to conceive of ophthalmologic problems in the broader light of systemic disorders, and his attitude toward original thought was that no observation or contribution can be too trivial to be worthy of recording.

A review of his forty-six published articles reveals three outstanding features. The first was his attempt to apply preventive medicine by making specialists and general practitioners aware of disease before it has made its inroads. In 1912 he pleaded for departments in hospitals to be devoted to preventive medicine. In 1919 he stated that the early diagnosis of syphilitic involvement of the optic pathways is possible only through routine periodic ophthalmologic examinations of every patient with syphilis from the very beginning of the infection throughout life. From 1912 until recently he labored to be able to detect glaucoma not only in its early phases but even in the preglaucoma stage, through studies on ocular drainage and pupillographic examinations. To him is due much credit for making even the layman conscious of glaucoma.

The second feature was his aptitude for judging valuable contributions of others from abroad, and he was the first to utilize them here at home. In 1919 he reported the first case of bilateral glioma of the retina in which the nonenucleated eye was treated with radium and recovered useful vision. The patient had been under observation for over ten years. In 1929, after visiting Gonin abroad, he performed the first Gonin operation for retinal detachment done in the United States, at the Herman Knapp Memorial Eye Hospital. In 1934, through his efforts, the first checking station for tonometers was established at the same hospital.

The third feature was his early grasp of anaphylaxis and his correct predictions of its possibilities. In 1914 he was awarded the Lucien Howe prize of the Medical Society of the State of New York for a contribution to the experimental study of anaphylaxis. He correctly evaluated Elschnig's work in the production of experimental anaphylaxis with tissues of the eye as antigens, and stated [1]:

"The facts regarding the hypersensitization of the entire organism and of the eye by the use of extracts from uvea are bringing us nearer to one of the most vital questions in ophthalmology vis:-sympathetic ophthalmia."

He also predicted [1] (page 27), "I should not consider [it] improbable that anaphylaxis may be some day a routine and a very valuable method of differential diagnosis."

Genius, like Egypt's monarch timely wise,
Erects its own memorial ere it dies.

As long as society values progressive practical research, untiring enthusiasm in helping one's fellowman and constant striving for truth, Dr. Mark J. Schoenberg will be with us.

ISADORE GIVNER, M.D.

1. Schoenberg, M. J.: Contribution to Experimental Study of Ocular Anaphylaxis, Ophthalmology **11**:4, 1914.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Conjunctiva

MENINGOCOCCIC CONJUNCTIVITIS. R. D. REID and L. H. BRONSTEIN, J. A. M. A. **124:** 703 (March 11) 1944.

A child of 2 years had purulent conjunctivitis of the right eye. A smear showed numerous intracellular and extracellular gram-negative diplococci. The condition of the eye grew worse. General examination revealed nothing of significance except for a temperature of 99.6 F. Cultures of material made by rubbing the fresh swab of exudate on the surface of a chocolate agar plate yielded an abundant growth of oxidase-positive, gram-negative diplococci. The organisms were agglutinated with antimeningococcus serum type I and failed to agglutinate with antimeningococcus serum type II or III or with antigonococcus serum. With sulfathiazole therapy and irrigations of the eye with a solution of boric acid, there was no longer any purulent discharge on the third day, and the child was dismissed from the hospital.

The authors conclude that the diagnosis of meningococcic conjunctivitis should be suspected in cases in which gram-negative intracellular diplococci are seen in smears of pus and no obvious source of the infection is discernible.

W. ZENTMAYER.

UNILATERAL FOLLICULAR CONJUNCTIVITIS DUE TO MOLLUSCUM CONTAGIOSUM. J. A. MAGNUS, Brit. J. Ophth. **28:** 245 (April) 1944.

A woman aged 53 had acute catarrhal conjunctivitis with superficial keratitis of the right eye, which had developed after influenza. Examination of the nose, throat and sinuses revealed nothing abnormal. A culture was sterile. At the end of a month there was temporary improvement. A relapse occurred, and follicles developed in the conjunctiva. It was not until three months after the first examination that several umbilicated, small tumors were noticed on both lids. Two of these encroached on the lid margins. The tumors were removed, and the growths at the margin of the lid were destroyed by electrocautery. A piece of the conjunctiva was excised. The histologic examination confirmed the diagnosis of follicular conjunctivitis molluscum contagiosum.

The article is illustrated.

W. ZENTMAYER.

Cornea and Sclera

FURTHER STUDIES ON RELATIONSHIP OF CORNEAL VASCULARIZATION TO RIBOFLAVIN DEFICIENCY. J. F. MCCREARY, J. V. V. NICHOLLS and F. F. TISDALL, Canadian M. A. J. **51:** 106 (Aug.) 1944.

McCreary and his associates report on studies to determine whether or not examination with the slit lamp and the photographic procedure give comparable data and to throw further light on the effect of riboflavin on corneal vascularization and symptoms of ocular fatigue. The results obtained from photographing the corneoscleral junction with the ophthalmic camera and the results of examination with a slit lamp are not significantly different. A study to demonstrate the effect of riboflavin on corneal vascularization was carried out, using both photography and examination with the slit lamp. The subjects studied were 41 students in the photographic division of the Canadian Air Force who had been provided with a ration containing when served 2.9 mg. of riboflavin per day for a period of one year. Approximately one-half the subjects were given a supplement of 3.3 mg. of riboflavin three times a day for two months, and the other half received placebos. There was no consistent change in corneal vascularization in either the treated subjects or the controls. The instillation of a simple irritant in the conjunctival sac caused collapsed, afunctional blood vessels in the cornea, transitional zone and conjunctiva to become engorged. This study seems to show that a uniform vascularization of the periphery of the cornea is not a safe basis for a diagnosis of riboflavin deficiency existing at the time of examination. Such a lesion may be due to riboflavin deficiency, but the deficiency could have occurred at any time prior to the examinations. Also, these blood vessels could have been reactivated by some cause other than lack of riboflavin.

J. A. M. A. (W. ZENTMAYER)

General

TREATMENT OF ASTHENOPIA–NONPATHOLOGIC AND NONREFRACTIVE IN ORIGIN. R. H. PINO and G. L. HULTIN, Am. J. Ophth. **27:** 520. (May) 1944.

Pino and Hultin give the data on 125 patients with asthenopia who were not relieved by glasses or other means. The ages ranged from 12 to 54. They feel that orthoptic training may be helpful

in ways over and above the more definite procedures for which such exercises are more commonly used.

W. S. REESE.

General Diseases

CONGENITAL CATARACT AND OTHER ANOMALIES FOLLOWING GERMAN MEASLES IN THE MOTHER. A. B. REESE, Am. J. Ophth. **27:** 483 (May) 1944.

Reese reports 3 cases of congenital cataract and congenital heart lesions in infants whose mothers had contracted rubella within the first month of pregnancy. He refers to similar cases reported in Australia after a severe epidemic of rubella.

W. S. REESE.

ALLERGIC REACTION OF TEARS OF TUBERCULOUS PATIENTS. C. GARBINO, Arch. de oftal. de Buenos Aires **18:** 617 (Nov.) 1943.

Observations were made on children, including 20 healthy children, 22 with phlyctenular hyperergic or nonallergic keratoconjunctivitis and 10 children with tuberculosis.

The tears were injected, using the Mantoux technic for inoculation with tuberculin. The observations were divided as follows: Tears of children with hyperergic phlyctenular keratoconjunctivitis were inoculated into children with the same type of conjunctivitis, into children with the nonallergic phlyctenular type, into children with the allergic nonphlyctenular type and into healthy children.

The control tests consisted in injection of tears of healthy children into allergic children, with phlyctenular or nonphlyctenular conjunctivitis and into nonallergic children with phlyctenular and nonphlyctenular keratoconjunctivitis.

The results were 100 per cent positive in the first group, 100 per cent negative in the second group and 100 per cent negative in the control groups. The author states that these experiments tend to confirm the pathogenic concept of Weekers in regard to the cause of phlyctenular keratoconjunctivitis.

H. F. CARRASQUILLO.

Injuries

ERRORS IN THE ROENTGENOGRAPHIC DIAGNOSIS OF FOREIGN BODIES IN THE EYE. J. A. SENA, Arch. de oftal. de Buenos Aires **18:** 622 (Nov.) 1943.

In the diagnosis of an intraocular foreign body one must consider the probable and the true evidence. The first is furnished by the history, the study of the wound and the reactive phenomena. The only true sign is the visualization of the foreign body inside the eye, and this, unfortunately, is possible in only 5 per cent of cases. For this reason the use of the roentgenogram is of the utmost importance in the diagnosis, although not altogether infallible. Even with a negative roentgenogram, the existence of an intraocular foreign body cannot be absolutely excluded. The author has been able to show roentgenographically a steel particle weighing only 1.6 mg. and a copper fragment weighing 0.97 mg.

The following rules should be observed for exactness of roentgenographic studies: the use of soft rays produced by a tube of fine focus; the avoidance as much as possible of osseous structures, and the proper immobilization of the head and eye to be examined, in order to make visible in the film the contour of the eyeball, or in some way to make possible an estimation of its contour.

Sena recommends the Sweet technic and the geometric method as the best procedure. The only objection to its use is the necessity for a special apparatus. Attention is called to the possible fallacy of the roentgenographic diagnosis as proved in observations in 3 cases of his own. In 1 case a defect in the roentgenologic technic indicated the presence of a foreign body, while the clinical investigation gave negative evidence; in the second case the roentgenogram did not show the foreign body, while the clinical examination demonstrated its presence, and in the third case the roentgenographic examination showed that the foreign body was extraocular, while the clinical findings indicated that it was intraocular.

H. F. CARRASQUILLO.

Ocular Muscles

EXOPHTHALMIC OPHTHALMOPLEGIA. I. D. FAGIN, R. W. PAGEL and H. H. SAND, Am. J. Ophth. **27:** 504 (May) 1944.

The authors give the following summary:

"1. The clinical history and findings of postmortem examination of a patient with exophthalmic ophthalmoplegia are reported.

2. The syndrome of exophthalmic ophthalmoplegia is briefly reviewed."

W. S. REESE.

DUTIES AND TRAINING OF AN ORTHOPTIC TECHNICIAN. W. B. LANCASTER, Am. J. Ophth. **27:** 515 (May) 1944.

Lancaster feels that training of orthoptic technicians has been deficient in psychology, the art of teaching, the laws of learning and the methods of habit formation of conditioned reflexes.

W. S. REESE.

RECESSION OF THE TROCHLEA FOR REDUCING THE ACTION OF THE SUPERIOR OBLIQUE MUSCLE. W. L. HUGHES, Am. J. Ophth. **27:** 1123 (Oct.) 1944.

Hughes reports 8 cases of recession of the trochlea for overaction of the superior oblique muscle. He states that the procedure is simple, effective and safe and can be done with local anesthesia.

W. S. REESE.

DIVERGENCE EXCESS CONSIDERED AS AN ANOMALY OF THE POSTURAL TONUS OF THE MUSCULAR APPARATUS. A. POSNER, Am. J. Ophth. **27:** 1136 (Oct.) 1944.

Posner gives the following summary:

"1. A case of divergence excess combined with an esophoria is reported.

"2. The prevailing theories regarding the nature of divergence excess are discussed.

"3. It is shown that divergence excess is due neither to excessive stimulation of the divergence center nor to an anomalous position of rest dependent on anatomic factors.

"4. It is proposed to regard divergence excess as an anomaly of the postural tonus of the extrinsic muscular apparatus in an atavistic sense.

"5. According to this view, the eyes are capable of maintaining one of two alternative postures: a more primitive divergent one, or a more recently acquired parallel one, depending on the degree of cortical control which is being exercised."

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

THE EXOPHTHALMOS OF HYPERTHYROIDISM. J. H. MULVANY, Am. J. Ophth. **27:** 589 (June) 1944.

This is part I of a long article on exophthalmos based on the Hunterian lecture delivered in February 1939. It deals with differentiation on the basis of the mechanism, pathologic picture, symptoms and treatment of two varieties of exophthalmos associated with hyperthyroidism.

W. S. REESE.

PROGRESSIVE EXOPHTHALMOS IN TOXIC DISEASE OF THYROID GLAND: REVIEW OF RECENT LITERATURE, WITH REPORT OF CASE OF PROGRESSIVE POST-THYROIDECTOMY PROPTOSIS IN A SIX YEAR OLD NEGRO GIRL. G. M. HAIK, Arch. Surg. **48:** 214 (March) 1944.

Haik states that treatment of exophthalmos associated with hyperthyroidism, particularly the variety which becomes progressive after thyroidectomy, has been unsatisfactory. Thyroidectomy frequently seems to aggravate it. Recent studies indicate that the cause of exophthalmos accompanying hyperthyroidism is local edema, sometimes associated with hypertrophy of the extraocular muscles. The author reports a case of progressive post-thyroidectomy exophthalmos which he observed in a Negro girl aged 6 years. The case is of interest because toxic thyroid disease in very young children is unusual, and is even more unusual in young Negro children. The character of this patient's toxicity was evidenced by her extreme nervousness; her voracious appetite, with continued loss of weight; her constantly elevated pulse rate, respiratory rate and temperature; the crises which developed on two occasions, and the degree of damage to the liver demonstrated ante mortem by serial tests of function and confirmed by postmortem examination. The child's parents disregarded instructions given them when she was dismissed after the first operation. Ten months later irreparable damage to the eyes had occurred. The optic tissues removed at Naffziger's unroofing operation showed edematous changes, which were also present, though to a lesser degree, in the tissues removed post mortem. The case seems to fit into the "special ophthalmic type" of goiter described by Means and his associates. It may be that the use of radiation, rather than thyroidectomy, might have saved the child's eyes and ultimately her life. It is reasoned that with a less abrupt alteration in the endocrine balance induced by irradiation there is a greater chance for more gradual readjustment of the optic structures.

J. A. M. A. (W. ZENTMAYER)

The Pupil

A SIMPLE METHOD FOR THE EARLY DIAGNOSIS OF ABNORMALITIES OF THE PUPILLARY REACTION. H. J. STEARNS, Brit. J. Ophth. **28:** 275 (June) 1944.

The procedure suggested by Stern is to throw a pinpoint light on the eye with the slit lamp in such a way that it just enters the pupil near the margin of the iris; this causes a light reaction, and the pupil contracts. This means that the margin of the iris moves toward the center of the pupil and prevents the light pencil from entering the pupil. As no light now reaches the retina, the stimulus for the contraction of the pupil is no longer present; the pupil dilates and thus again allows the pencil of light to reach the retina. The whole mechanism starts anew. This artificial hippus continues regularly in the normal eye so long as the light pencil enters the pupil. Physiologic differences, such as age, color of the iris and errors of refraction, have no influence on the reaction. Elderly persons and persons with dark irises show less extensive movements, but in every normal person the reaction is easily produced. The pupil contracts and dilates ten times in seven to eight seconds.

In cases of such conditions as latent syphilis and early tabes, with clinically normal pupillary reactions, the artificial hippus could not be produced with the slit lamp. Usually a good, if not extensive, contraction followed the first stimulus; then either the pupil did not dilate sufficiently to let the light pencil enter the eye, or a few irregular, sluggish contractions followed, after which the pupil finally remained immobile.

W. ZENTMAYER.

Physiologic Optics

A Reply to Certain Criticisms of Aniseikonia. W. B. Lancaster, Am. J. Ophth. **26:** 943 (Sept.) 1943.

Lancaster gives a brief presentation of the methods of measuring aniseikonia and of the principles involved and then considers the allegations and criticisms. A few cases are reported, with sufficient data that one may form one's own opinion.

W. Zentmayer.

Retina and Optic Nerve

A Case of Rupture of the Retinal Cyst Causing Retinal Detachment. C. D. Shapland, Brit. J. Ophth. **28:** 236 (April) 1944.

A soldier aged 28, seven weeks before examination of his eyes, had felt a sharp, shooting pain, lasting two or three minutes, in the region of the right temple and felt dizzy and faint.

In the right eye a flat detachment of the retina involved the macula. At the 7 to 9 o'clock position there was a prominent, thin-walled cyst with a delimiting band of retinochoroidal pigment disturbance. The wall of the cyst had evidently ruptured just behind the ora serrata, leaving a ragged dialysis of the retina. A diathermy operation was successful in replacement of the retina. Visual acuity, which had been 3/60, improved to 6/24, and a wide reentering angle in the field flattened out.

The article is illustrated.

W. Zentmayer.

Trachoma

Trachoma. W. G. Forster and J. R. McGibony, Am. J. Ophth. **27:** 1107 (Oct.) 1944.

In a study of trachoma among the North American Indians, it was found that there was only a slight decrease in the incidence of the disease between 1912 and 1938, but following the introduction of the sulfonamide compounds, in 1939, there was a notable reduction in the incidence.

Pannus must be present if the diagnosis of trachoma is to be made.

Local therapy with sulfonamide compounds has not been encouraging.

Over 90 per cent of the patients were cured after one course of treatment if an adequate dose of sulfanilamide was employed.

Conditions not responding to sulfanilamide therapy are probably not trachomatous.

W. S. Reese.

Tumors

Leiomyoma of the Iris. W. T. Davis, E. Sheppard and W. J. Romejko, Am. J. Ophth. **27:** 467 (May) 1944.

The authors report a case of leiomyoma and suggest excision, rather than enucleation, of such a tumor because it does not invade surrounding structures or the filtration angle.

W. S. Reese.

Extradural Diploic Epidermoids Producing Unilateral Exophthalmos. E. H. Thornhill and B. Anderson, Am. J. Ophth. **27:** 477 (May) 1944.

Thornhill and Anderson give the following summary and draw the following conclusions:

"Extradural diploic epidermoids producing unilateral exophthalmos are not usually considered among the possibilities in the differential diagnosis of the causes of this condition. None the less, the condition may exist in areas adjacent to the orbit and may be the cause of exophthalmos. The possibility that unilateral exophthalmos is due to such a lesion can probably be proved or eliminated by X-ray studies once the possibility is recognized by the typical scalloping of the edges with marginal increase in bone density demonstrated through proper angulation and exposure.

"Complete removal and a cure was obtained in this case through the supra-orbital approach. It is doubtful whether this particular type of tumor ever occurs so far back in the orbit (the tumor arises from diploic spaces which are not present in the walls of the orbit) as to justify the transfrontal approach recommended by neurosurgeons."

W. S. Reese.

Epithelial Tumors of the Iris. M. K. Asbury, Am. J. Ophth. **27:** 1094 (Oct.) 1944.

Asbury discusses tumors of the iris and cites reported cases. From a detailed study of 4 cases she concludes that in spite of relatively benign histologic characteristics, these tumors all showed invasiveness and local destructiveness and were probably malignant in the same sense as is rodent ulcer of the skin (basal cell carcinoma).

W. S. Reese.

Vision

Suppression Amblyopia. H. S. Sugar, Am. J. Ophth. **27:** 469 (May) 1944.

Sugar draws the following conclusions:

"1. In disagreement with Peter's statement that amblyopia in squinting eyes is a symptom of squint and not its cause, suggestive evidence has been presented indicating that suppression and amblyopia precede the development of certain nonparetic squints and follows the development of other nonparetic squints and those of paretic origin.

"2. Anisometropia is the predominating associated factor in nonsquinting amblyopic subjects, whereas high isohyperopia is the most frequent factor in cases of amblyopia with convergent squint.

"3. Nonparetic squint probably requires suppression plus another factor for its development. This may be the accommodation-convergence relationship or simply the assumption of the dissociated position after suppression has been obtained in one eye. The age factor is obviously of considerable additional importance.

"4. Suppression amblyopia is a more accurate term than amblyopia ex anopsia, implying, as it does, an active rather than a passive process.

"5. The conclusions drawn may be of aid in adding links to the chain of evidence as to the cause of squint. Further clarification of the reason for normal muscle balance in a large number of amblyopic subjects, particularly a possible relationship to peripheral fusion, is necessary."

W. S. REESE.

Therapeutics

THE USE OF TYROTHRICIN, A BACTERIAL EXTRACT, IN THE TREATMENT OF MARGINAL ULCERS OF THE CORNEA. S. BLOOMFIELD, Am. J. Ophth. **27**: 500 (May) 1944.

Bloomfield reports 4 cases of resistant marginal ulcer in which tyrothricin was used, with gratifying results. The drug may be readily applied to the conjunctival sac in effective concentrations. It has been proved effective against gram-positive organisms.

W. S. REESE.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Pan-American Congress of Ophthalmology.—The program for the coming meeting of the Pan-American Congress of Ophthalmology, which is to be held in Montevideo, Uruguay, during the week of Nov. 25, 1945, is practically completed and will be published in the near future. As was done at the last congress, all papers will be in Spanish, Portuguese or English, with projected translations as the paper is being presented.

Any recognized ophthalmologist is eligible for membership. The annual fee is $5, payable to Dr. Conrad Berens, treasurer, 218 Second Avenue, New York 3. That fee entitles the member to receive gratis the official organ of the congress —*Ophthalmologia Ibero Americana*, which is a quarterly trilingual abstract journal.

Tentative travel arrangements are being made with the Pan-American Airways, as well as the American Express Company, but these cannot be concluded definitely until there has been a further turn in world affairs.

Society of the Kellogg Fellows of the Pan-American Congress of Ophthalmology.—The Society of the Kellogg Fellows of the Pan-American Congress of Ophthalmology was formed to help in bringing about a close relationship between the ophthalmologists of the Americas and to aid in raising the level of the theory, practice and teaching of ophthalmology in the Western Hemisphere. The society met in October 1944, adopted the statutes of the organization and elected the following officers: Harry S. Gradle, M.D., (United States), honorary president; Manoel da Silva, M.D. (Brazil), president; Olga Ferrer, M.D. (Cuba), secretary.

The membership of the society is now composed of twenty-eight fellows from the following eighteen countries: Bolivia, Brazil, Chile, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, Guatemala, Haiti, Honduras, Mexico, Nicaragua, Paraguay, Peru, Puerto Rico and Venezuela.

Address:—Olga Ferrer, secretary, Institute of Ophthalmology of the Presbyterian Hospital, 635 West One Hundred and Sixty-Fifth Street, New York.

William Thornwall Davis Postgraduate Course in Ocular Surgery, Pathology, Ocular Motility and Orthoptics.—The eighth annual William Thornwall Davis Postgraduate Course will be given at the George Washington University School of Medicine, Washington, D. C., May 28-June 2, 1945. The Army Institute of Pathology, directed by Col. J. E. Ash, Medical Corps, United States Army, will give the course in ocular pathology, as he has done in former years. The courses in ocular surgery, ocular motility and orthoptics will be given by members of the resident staff of the department of ophthalmology, under the direction of Dr. Ernest Sheppard, professor of ophthalmology. The course is limited to 30 registrants.

SOCIETY NEWS

Cancellation of Annual Meeting of Association for Research in Ophthalmology.—The Association for Research in Ophthalmology has canceled its 1945 meeting in cooperation with the war travel and convention program. Essayists are requested to reserve their manuscripts for a possible meeting in 1946, in conjunction with the annual sessions of the American Medical Association.

UNIVERSITY NEWS

Appointment for Dr. Ernest Sheppard.—Dr. Ernest Sheppard has recently been appointed as professor of ophthalmology of the George Washington University School of Medicine, to succeed the late Dr. William Thornwall Davis. This appointment was directed by the board of trustees of the university on Dec. 1, 1944.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

THOMAS H. JOHNSON, M.D., *Chairman*

WENDELL L. HUGHES, M.D., *Secretary*

Regular Meeting, Jan. 15, 1945

Pigmented Meningioma and Neuroepithelioma of the Optic Nerve: Report of a Case. DR. CHARLES A. PERERA.

An interpretation of this extraordinary case is difficult. The orbital tumor was distinct from the neuroepithelioma and appeared to arise in the sheath of the optic nerve. The hyalinized masses may represent massive hyalinization of capillaries. The presence of chromatophores in meningiomas has been reported, and the cells are known to occur in neurofibromatosis. Gliomas of the optic nerve stimulate glial proliferation and practically never recur after removal of the main tumor. Perhaps the neuroepithelioma in this case produced a chemical agent which passed forward into the sheath of the optic nerve and stimulated growth of the pigmented meningioma.

Trauma and Ocular Lesions: A Study in Reciprocal Relations. DR. PERCY FRIDENBERG.

Millions are engaged in activities involving violent physical exertion in industry and even in "heavy" sports, in which serious injury to the limbs and the viscera, the skull and the bones, is almost an everyday occurrence. Detachment of the retina as a result of such exertion is practically unknown. Direct injury, especially to an eye predisposed by high myopia and its attendant pathologic changes, is, on the other hand, a probable factor in the causation of retinal detachment.

DISCUSSION

DR. ARNOLD KNAPP: Dr. Fridenberg has brought out many interesting and suggestive points. As far as retinal detachment is concerned, I think that trauma is an insignificant etiologic factor. Of the 400 cases of retinal detachment in which I made careful examinations, there was direct trauma in 32 and indirect trauma in 20. In the latter group the defense mechanism of which Dr. Fridenberg speaks is the only explanation; that is, the eyes were predisposed to the development of a detachment.

DR. MORRIS DAVIDSON: Dr. Fridenberg has dealt with the subject in a comprehensive manner, but there is one thing in particular to which I should like to refer. First, the importance of the relation of trauma to lesions of the eye is not realized. Perhaps some figures may be of interest. Of every 1,000 persons with injury examined at the Department of Labor, 250 present a problem in causal relations, and 125, a problem of the kind that Dr. Fridenberg discussed, namely, optic nerve atrophy, retinal detachment, interstitial keratitis, glaucoma and similar conditions. This problem will be a more important one in the future because of a greater number of workmen's compensation cases to be dealt with after the war. There is another important aspect with which some may not be familiar. A new amendment was passed at the last session of the New York state legislature which changes the emphasis of the topic that Dr. Fridenberg discussed. As you know, in the past the insurance carrier paid for the loss of an injured eye regardless of a preexisting defect. Now, in order to create more jobs by stimulating employers to take on defective persons and war veterans, the New York state law says that this loss will be apportioned between two parties: The insurance company will pay a part of the compensation allowed, and from a special fund to be created by the state the rest will be paid. The insurance company will pay for the loss resulting from trauma, and the state fund will be used to pay for the predisposition factor; so the emphasis on the causal relation will be less. The insurance company will be concerned less with the causal relation and more with finding out how much of the whole compensation it is to pay. The problem of the physician will be to appear on the witness stand and state, for example, whether the trauma accounts for 20 per cent of the loss and predisposition for 80 per cent, or whether the trauma accounts for 30 per cent of the loss and predisposition for 70 per cent. The problem will be to weigh the importance of the two roles, which is as difficult as the aspect dealt with by Dr. Fridenberg.

DR. PERCY FRIDENBERG: I want to mention a point I passed over when I was discussing the defense mechanism. This mechanism concerns not only the eye but the whole body, and the factors which bring about the defense of the eye will also produce tension of the abdominal muscles, forced expirations and all the accompaniments of heavy work. A number of cases were mentioned in which detachment of the retina was noted after coughing, sneezing or vomiting. Authors state that such actions could not cause

detachment; but they are accompanied with exactly the same reflexes in the defense mechanism of the eye. The grip on the globe is to protect it from sudden opening, like the protection provided by a pressure bandage. The lid has been called the protective dressing of the eye, and it might also be called its guardian angel.

Value of Cultures Before Operations for Cataract. Dr. John H. Dunnington and Dr. D. Locatcher-Khorazo.

This article will be published in a later issue of the Archives.

Relative Effects of Thiouracil, Iodine and Subtotal Thyroidectomy on the Exophthalmos of Hyperthyroidism. Dr. M. Noel Stow, Baltimore.

This study, of which the present paper is a preliminary report, is summarized as follows:

The comparative effects of thiouracil on a series of 10 patients, of iodine on 12 patients and of subtotal thyroidectomy on 10 patients, all with established hyperthyroidism, were studied.

The data obtained in this investigation were tabulated with special reference to the ophthalmopathy of exophthalmos and the associated widening of the palpebral fissures.

An attempt was made to correlate the normal with the abnormal hormonal activity of the thyroid and the sequential established pathologic states with reference to their possible alteration, especially in their ocular manifestations, by means of the therapeutic adjuncts of thiouracil, iodine and subtotal thyroidectomy.

Although the total number of cases available for study was not large enough to constitute a true biostatistical sampling, it appears (*a*) that the administration of thiouracil not only does not effect improvement in the exophthalmos of hyperthyroidism but tends to permit the exophthalmos to progress unchecked or to accelerate it or both; (*b*) that the preoperative administration of iodine in cases of recent onset tends either to effect no change in the exophthalmos or to bring about some improvement in it, and (*c*) that subtotal thyroidectomy most frequently results in progression of or no change in the proptosis.

The exophthalmos of hyperthyroidism continues to present a most difficult therapeutic problem. The administration of iodine is effective in some cases, but they must be carefully selected. The more radical procedure of subtotal thyroidectomy is frequently indicated when medical treatment is ineffective. Thiouracil, on the contrary, is most useful in alleviating the general symptoms; unfortunately, it may prove to be without benefit with regard to the ocular manifestations of hyperthyroidism. It is urged, therefore, that whether or not ocular symptoms are present, repeated complete and careful ophthalmologic examinations be done routinely on all patients with hyperthyroidism to whom thiouracil is administered.

DISCUSSION

Dr. David P. Barr: There seems to be no question that Dr. Stow has demonstrated an increase in the protrusion of the eyeballs in a number of cases following treatment with thiouracil and that this change is comparable in extent to the increase which is observed in some cases after subtotal thyroidectomy. Especially interesting is his demonstration that the protrusion of the eyeballs in some cases may be slightly diminished by the administration of iodine. These observations are substantiated in part by the recent unpublished observations of Dr. B. M. Dobyns, at the Mayo Clinic. Dr. Dobyns has permitted me to mention them. He studied 223 patients treated by subtotal thyroidectomy, making approximately 10,000 separate measurements of exophthalmos in the postoperative period. He was able to show in almost all his patients an increase in the prominence of the eyes following thyroidectomy. He was unable to show that iodine increased exophthalmos. Like Dr. Stow, he found that the exophthalmos became greater after the use of thiouracil. In 1 case he demonstrated an increase in protrusion of 4 mm. following the use of this drug.

My associates and I at New York Hospital have been interested in the use of thiouracil for about sixteen months, and during that time we have used the drug in the treatment of 85 patients. Sixty of them have been treated long enough to permit one to judge the full therapeutic effect. Some of them have received thiouracil continuously for nine months and most of them for six months or longer. Early in our studies we heard from Dr. R. H. Williams, of Boston, that malignant exophthalmos, similar to the form not infrequently observed after thyroidectomy, had developed in a patient treated with thiouracil. We therefore were prepared to believe that we might produce serious protrusion of the eyeballs with this active drug. Consequently, we made careful measurements with the Hertel exophthalmometer before and at intervals after the use of the drug. I am sure that the accuracy of our measurements is not comparable to that of Dr. Stow's, for they were carried out by a number of persons. We did, however, observe increases as high as 3 mm. in 1 patient and as high as 2 mm. in several others. No patient exhibited a diminution in the protrusion of the eyeballs. We did not measure the width of the palpebral fissure. We did, however, observe that many of the patients treated with thiouracil had diminished lid spasm and lid lag, so that, while the actual protrusion was not ameliorated, exophthalmos appeared to be lessened. We treated 3 patients who would be regarded, I think, as having malignant exophthalmos. In none of them was there a

measurable increase in the degree of protrusion. In 2 patients there was no improvement in function or in clinical symptoms. In the third patient the degree of lid spasm had diminished so much that all troublesome symptoms disappeared.

Of course it is impossible to generalize from such scant evidence; but it is our impression at present that thiouracil does not lessen the protrusion of the eyeballs, that in some cases it increases it but that in such cases the increase is so slight that one cannot regard it as dangerous to the eyes, even in cases in which there are beginning symptoms of malignant exophthalmos.

Dr. G. K. Smelser (by invitation): It is difficult for me, whose experience with exophthalmos lies entirely in experimental work with guinea pigs, to follow Dr. Stow's paper and Dr. Barr's discussion. Dr. Stow's paper is particularly timely because, as you know, thiouracil is a new drug which is being used widely, and not only at Cornell, the New York Hospital and the Presbyterian Hospital. Many patients are being treated by medical men who are less experienced in the examination of eyes than are ophthalmologists, and they sometimes are surprised that the ocular symptoms are not improved. It is interesting that this report confirms the extrathyroid origin of exophthalmos. At one time it was thought that exophthalmos was one of the characteristics of hyperthyroidism and that the secretion of the thyroid was responsible for the exophthalmos; I think that belief is no longer held. Apparently, one must look to some gland or structure outside the thyroid for the cause of the protrusion of the globe and of the other symptoms of exophthalmos, which go hand in hand with those referable to the thyroid and are the result of the same malfunction or have the same etiologic factor. Apparently, on the basis of laboratory experiments which my associates and I have carried out, the pituitary does seem to be involved. The observations of Dr. Stow tend to show that something outside the thyroid is responsible for the protrusion, for he has demonstrated that with the shift toward normal of the serum cholesterol, the body weight and the metabolism, all the symptoms have been abolished or alleviated except the exophthalmos.

Several questions come to my mind. First, Dr. Stow reported that in some of the thiouracil-treated patients there was no change in the exophthalmos, or the eye protruded even more, but that the spasm of the lid or the width of the palpebral fissure did not change. What is the controlling factor in the lid spasm and the width of the palpebral fissure? I shall go back to my guinea pigs, for it seems that the palpebral fissure may be controlled by two mechanisms. In rodents with hyperthyroidism the palpebral fissure is controlled largely by the sympathetic fibers. The hyperthyroidism could, I think, be cured with thiouracil, for the basal metabolic rate decreases and the toxic manifestations are lessened. They should also decrease with the use of iodine. However, in a number of experiments the width of the palpebral fissure did not decrease, even though involvement of the sympathetic fibers was ruled out. The lid lag was due to a forward movement of the globe. I do not know whether this could be the cause of the widened palpebral fissure in the patients with subtotal thyroidectomy.

I think Dr. Stow's observation as to the effect of iodine is an original contribution. In a review of the literature, one finds almost anything; one author says the drug is of benefit and another that it is not, and one cannot make up one's mind whether iodine in the form of compound solution of iodine U.S.P. is of value in the treatment of exophthalmos. When, in our experiments with guinea pigs, we produce exophthalmos with pituitary extract, we can give the animals any quantity of iodides without a decrease in the exophthalmos. Apparently, from these experiments one would judge that the iodine molecule does not interfere with the action of the pituitary extract when injected parenterally. Then (this is a working hypothesis), if this is the case, and the iodine given the patients in Dr. Stow's series had a beneficial effect on the exophthalmos, it must have been due to a release of pituitary hormone from the pituitary gland itself, it being always assumed that the pituitary is the basic cause of the exophthalmos.

Another suggestion might be made. A number of years ago Moran, in an interesting paper read at Atlantic City, N. J., suggested that part of the exophthalmos of hyperthyroidism was really sympathetic in origin and was due to the palpebral muscle pulling back on the orbital septum and thus exerting pressure on the retrobulbar tissues, which pushed the globe forward. On this basis, the correction, or the decrease in exophthalmos, which Dr. Stow found associated with hyperthyroidism, might be due to the relaxation of the sympathetic stimulus.

The mechanism of the effect of iodine and of thiouracil on the exophthalmos is, I think, one of the most interesting and important phases of the work on exophthalmos. Certainly, the problem is a great one. The help that is to be obtained is now almost entirely in the clinical field, and a good example of this has been given by Dr. Stow.

Dr. Isadore Givner: I should like to make two observations. After listening to this paper, one may feel that the process of exophthalmos is probably not a reversible one, since thiouracil, iodine in most cases, and thyroidectomy produce no reduction in the exophthalmos. I should like to cite a case which will leave one a little more optimistic. A woman who had been operated on for exophthalmic goiter noticed an increase in the exophthalmos following thyroidectomy. This amount became stationary. The patient subse-

quently became pregnant, and the question came up as to whether the pregnancy should be allowed to go on to term. It was decided to let it go through without interruption. After she had been pregnant four months, the exophthalmos began to show improvement and continued to do so up to the time the baby was born. Measurements showed that the exophthalmos had receded 4 mm., demonstrating the fact that the changes in muscle which are responsible for exophthalmos are reversible. Interestingly, the baby was born with exophthalmos, which persisted until the child was 3 months old. The child is now 3 years old, and the mother's exophthalmos has stayed at the lower level. This brings up the question as to what happened during pregnancy that unfavorably influenced the exophthalmos. Did the baby's thyroid or thymus act for the mother, or were there certain hormonal changes which occurred either at the placental barrier or in the mother which produced changes in the muscles of the eyes? I am not in a position to answer these questions, but they suggest material for investigative work.

Dr. Otto Lowenstein and I studied 22 cases of exophthalmic goiter by pupillography, and in 21 of these there was a characteristic pupillographic curve, namely, a redilation block, which was interpreted as indicating a central sympathetic lesion, probably in the hypothalamus. This fits in well with the theoretic discussion presented this evening and gives tangible evidence of central disturbance.

Dr. M. Noel Stow: I wish to thank Dr. Barr and Dr. Smelser for discussing the paper. I have nothing to add to their discussion. In answer to Dr. Smelser's question as to the cause of the widened palpebral fissure in hyperthyroidism, the theory that is most plausible to me is that of sympathetic stimulation; this hypothesis is based on the accepted theory that in hyperthyroidism the threshold of the sympathetic nervous system is lowered. I think this holds true in the early stage of exophthalmos, but there is more than mere stimulation of the sympathetic system, particularly when the condition is of some duration. In fact, it seems to me that when the hyperthyroidism is reduced, whether by thiouracil, iodine or operation, there will no longer be pronounced sympathetic stimulation; but if the process has gone on long enough, the eyes become so proptosed that it is as much a mechanical as a sympathetic mechanism, or even more. In every case in which there was widening of the palpebral fissures there was a measurable exophthalmos. I am unable to answer the question of Dr. Givner with reference to the patient who had a decrease in her proptosis during pregnancy. I know that similar cases have been reported in the literature, but I am unable to explain the exact mechanism.

I do not wish to leave the impression that thiouracil is not a valuable drug. I believe that it is one of the better drugs and that in the future it may be found to be the best one, but I believe that when it is given in cases of exophthalmos the eyes should be followed very closely. In this series, which was small, there was no case of malignant exophthalmos and none of corneal involvement, and there was only moderate conjunctival chemosis in the worst cases. The protrusion was pronounced in only 1 case; in others the changes were slight. However, one should follow the ophthalmologic signs in the patients treated with thiouracil thoroughly enough and long enough to know more about the effect of the drug. Dr. Lahey believes that the best combination for preoperative preparation is thiouracil and iodine. It has been found that in a patient who has been prepared for operation with thiouracil there is an increased vascularity of the thyroid gland and that bleeding at the time of operation is most troublesome, but Lahey has given a verbal statement to another physician, who gave it to me, that in his hands administration of thiouracil for about three weeks, then discontinuance of the drug for one week, followed by administration of iodine is the most satisfactory preparation for operation he has thus far found.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. John Foster, 45 Park Sq., Leeds.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of South Africa

President: Dr. A. W. Sichel, National Mutual Bldg., Church Square, Cape Town.
Secretary: Dr. J. K. de Kock, Groote Kerk Bldg., 32 Parliament St., Cape Town.

Ophthalmological Society of the United Kingdom

President: Mr. Charles B. Goulden, 89 Harley St., London.
Secretary: Mr. Frank W. Law, 30 Devonshire Pl., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

São Paulo Society of Ophthalmology

President: Prof. Moacyr E. Alvaro, Consolação 1151, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santo Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

Scottish Ophthalmological Club

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

Sociedad Argentina de Oftalmologia

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

Sociedad Oftalmologia del Litoral, Rosario (Argentina)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Arturo Etchemendigaray, Villa Constitución, Santa Fé.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

Sociedade de Oftalmologia de Minas Gerais

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennio Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

Sociedade de Oftalmologia e Otorrinolaringologia de Rio Grande do Sul

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

Sociedade de Ophthalmologia e Oto-Rhino-Laryngologia da Bahia

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

Società Oftalmologica Italiana

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 Ill tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
In compliance with the request of the Office of Defense Transportation and in the interest of the national war effort a meeting will not be held in 1945.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

Association for Research in Ophthalmology, Inc.

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

Canadian Medical Association, Section on Ophthalmology

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Dr. R. Evatt Mathers, 34½ Morris St., Halifax, N. S.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Central Illinois Society of Ophthalmology and Otolaryngology

President: Dr. Watson Gailey, 1000 N. Main St., Bloomington, Ill.
Secretary-Treasurer: Dr. William F. Hubble, 861-867 Citizens Bldg., Decatur, Ill.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. James H. Mathews, 1317 Marion St., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. L. D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Harold H. Heuser, 207 Davidson Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

Sioux Valley Eye and Ear Academy

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

Southwestern Academy of Eye, Ear, Nose and Throat

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

Southwestern Michigan Triological Society

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

Arkansas State Medical Society, Eye, Ear, Nose and Throat Section

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

Colorado Ophthalmological Society

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

Eye, Ear, Nose and Throat Club of Georgia

President: William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Kansas State Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. W. D. Pittman, Pratt.
Secretary: Dr. Louis R. Haas, 902 N. Broadway, Pittsburg.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Val. H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N. St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. George P. Meyer, 410 Haddon Ave., Camden.
Secretary: Dr. John P. Brennan, 429 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. R. B. Maw, 699 E. South Temple, Salt Lake City.
Secretary-Treasurer: Dr. Charles Ruggeri Jr., 1120 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.

Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.

Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.

Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.

Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.

Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.

Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.

Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

BIRMINGHAM EYE, EAR, NOSE AND THROAT CLUB

President: Each member, in alphabetical order.

Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.

Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.

Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn 16.

Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.

Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.

Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.

Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.

Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Samuel J. Meyer, 58 E. Washington St., Chicago 2.

Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2.

Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

CINCINNATI GENERAL HOSPITAL OPHTHALMOLOGY STAFF

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.

Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.

Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. Shandor Monson, 1621 Euclid Ave., Cleveland.

Secretary: Dr. Carl Ellenberger, 14805 Detroit Ave., Cleveland.

Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.

Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.

Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Erwin W. Troutman, 21 E. State St., Columbus, Ohio.

Secretary-Treasurer: Dr. T. Rees Williams, 380 E. Town St., Columbus 15, Ohio.

Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. C. B. Collins, 704 Medical Professional Bldg., Corpus Christi, Texas.

Secretary: Dr. L. W. O. Janssen, 710 Medical Professional Bldg., Corpus Christi, Texas.

Time: 6:30 p. m., third Tuesday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.

Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.

Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.

Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.

Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.

Secretary: Dr. Cecil W. Lepard, 1025 David Whitney Bldg., Detroit 26.

Place: Club rooms of Wayne County Medical Society.

Time: First Wednesday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.

Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit 26.

Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.

Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.

Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.

Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.

Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.

Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.

Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.

Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Dennis Smith, 623 Security Bldg., Long Beach 2, Calif.

Secretary-Treasurer: Dr. Robert Null, 710 Security Bldg., Long Beach 2, Calif.

Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.

Secretary-Treasurer. Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.

Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.

Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.

Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Leon H. Guerin, 324 E. Wisconsin Ave., Milwaukee 2.

Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.

Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Thomas H. Johnson, 30 W. 59th St., New York.
Secretary: Dr. Wendell L. Hughes, 131 Fulton Ave., Hempstead, N. Y.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Milton Berliner, 57 W. 57th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Secretary: Dr. Robert J. Billings, 509 Liberty Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Isaac B. High, 326 N. 5th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.
Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.

Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.

Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.

Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.

Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.

Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.

Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.

Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.

Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.

Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.

Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. L. C. Ravin, 316 Michigan St., Toledo 2, Ohio.

Secretary: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo, Ohio.

Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.

Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.

Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Harold M. Downey, 1740 M St. N. W., Washington, D. C.

Secretary-Treasurer: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.

Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.

Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.

Place: Office of chairman. Time: Last Tuesday of each month from October to May.